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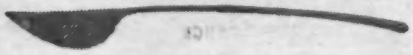

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TABLE OF CONTENTS

ORIGINAL ARTICLES: Page

1. Subdural Haematoma—V. Balasubramanian and B. Ramamurthi 1
2. Cirrhosis of the Liver with Special Reference to Intravenous Liver Extract Therapy: A Study of 35 Cases—Akbarali, R. P. Mehrotra, M. M. Arora and D. P. Mukerji ... 7
3. Prognostic Factors in Tuberculous Meningitis—S. S. Misra and B. K. Khanna 13

PRACTITIONERS' SERIES:

Chronic Pulmonary Emphysema—H. Ganguli ... 18

CASE NOTES:

1. Lymphadenopathy in a Case of Indian Kala-azar—P. C. Sen Gupta and Anjali Chatterji ... 21
2. Multiple Pseudocystic Tuberculosis of Bone in an Adult—K. S. Bose 22
3. Osteogenesis Imperfecta Tarda—S. R. Jain and K. N. Sanghvi 24

CASE NOTES—Contd. Page

4. Late Onset of Friedreich's Ataxia—R. S. Wadia, N. G. Tabib and G. S. Mutalik 25

EDITORIAL:

Tuberculous Meningitis 26

PRESIDENT'S COLUMN:

Shortage of Doctors 28

GENERAL PRACTICE DIGEST 28

CURRENT MEDICAL LITERATURE 30

CURRENT TOPIC:

Public Health Education and Radiation Hazards—M. G. Candau 32

NOTES AND NEWS 35

CORRESPONDENCE 39

REVIEWS 40

SUPPLEMENT:

Branch Notes 41

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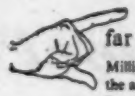

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Index to Advertisers

	Page
Antiseptic	viii
Alembic Chemical Works Co. Ltd.	xxviii
Allen & Hanburys Ltd.	xi
B. K. Shaw Industries (Private) Ltd.	x
Bengal Chemical & Pharm. Works Ltd.	Front Cover
Bengal Immunity Co. Ltd.	2nd cover
Boehringer-Knoll Private Ltd.	ix
Calcutta National Chemical Industries Private Limited	xxxiii
Corn Products Co. (I) Pvt. Ltd.	xiv
Crookes Laboratories Ltd.	xxxiv
Dr. S. Lahiri	3rd Cover
Dey's Medical Stores Private Ltd.	xxii
Dollar & Co.	xxxi
East India Pharm. Works Ltd.	xxiv
Eastern Drug Co. Ltd.	iv
Franco Indian Pharm. Pvt. Ltd.	vii, xiii
Farmavin Laboratories Pvt Ltd.	xx
G. D. Pharmaceuticals Pvt. Ltd.	xx
G. D. A. Chemicals Ltd. ,	iii
German Remedies & Trading Co. Private Limited	xxix
Glaxo Laboratories (I) Private Ltd.	xxxvi
Himalaya Drug Co.	xxvi
Hind Chemicals Ltd.	xxxiii
Ilford-Selo (India) Private Ltd.	xviii
Indian Health Institute & Labo. Ltd.	3rd Cover
International Chemical & Biological Institute Private Limited	xxix
J. & J. Dechane	xiv
John Wyeth & Brother Ltd	xxvii
Khandelwal Laboratories Private Ltd.	xvii
Khatau Valabhadas & Co.	xxxii
Lederle Laboratories (India) Pvt. Ltd.	i
May & Baker (India) Private Ltd.	vi, 3rd Cover
Navaratna Pharmaceutical Labo.	xxxiii
Neo-Pharma Private Ltd.	v, xxiii
Oriental Pharm. Industries Ltd.	viii
Phoenix Drug House Private Ltd.	xii
Rallis India Ltd.	xxx
Ranbaxy & Co. Private Ltd.	xxxiv
Raptakos, Brett & Co. Pvt. Ltd.	xxxi
Sarabhai Chemicals	xix
Scientific Publication Concern	xxxiii
Smith Kline & French (India) Ltd.	ii, xv, xxi
Smith Stanistreet & Co. Ltd.	xxxv
Stadmed Private Ltd.	x
Standard Pharmaceutical Works Ltd.	xvi
Suhrid Geigy Trading Private Ltd.	xxv
Unichem Laboratories	Back cover
Warner Lambert Pharmaceutical Co.	xii, xvi

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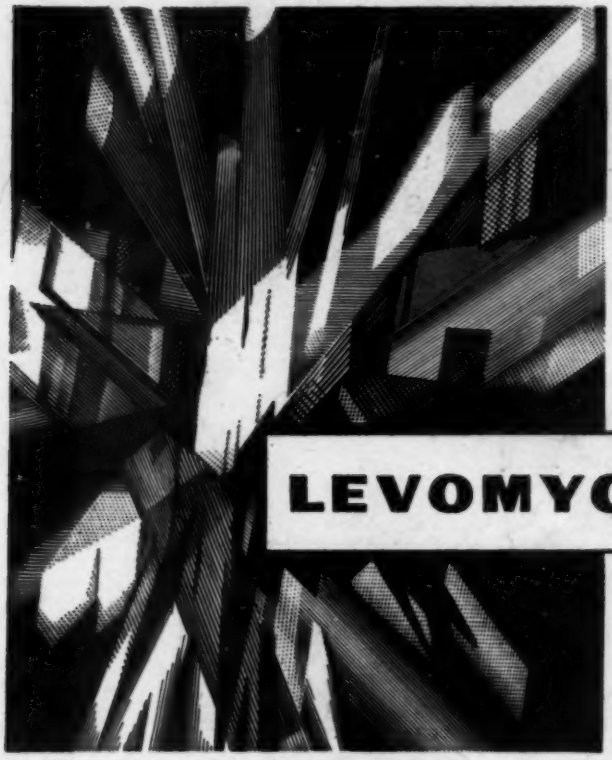

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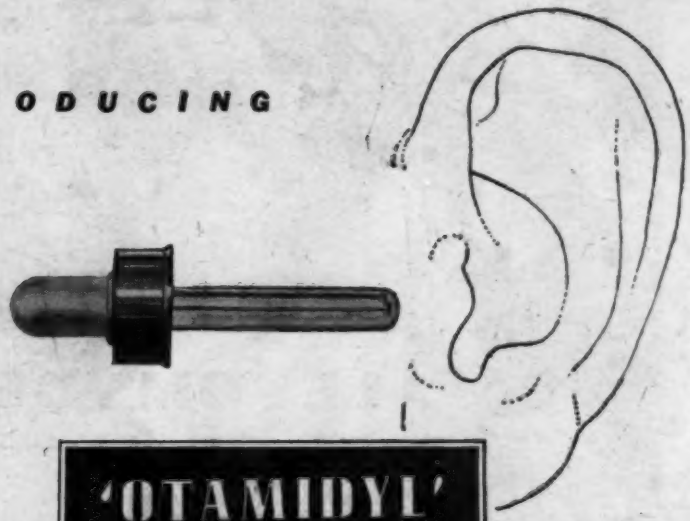

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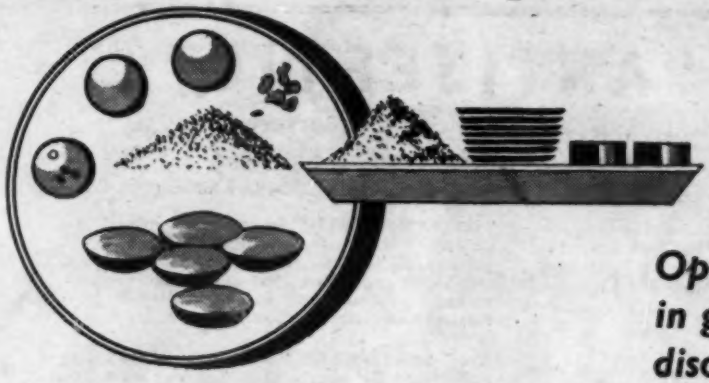

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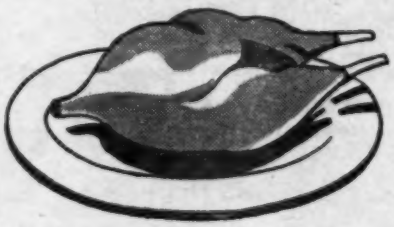

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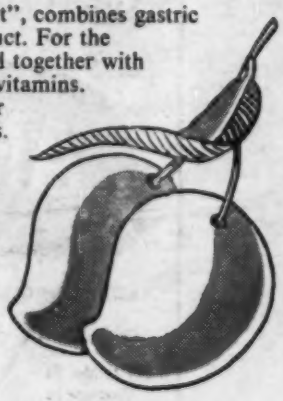

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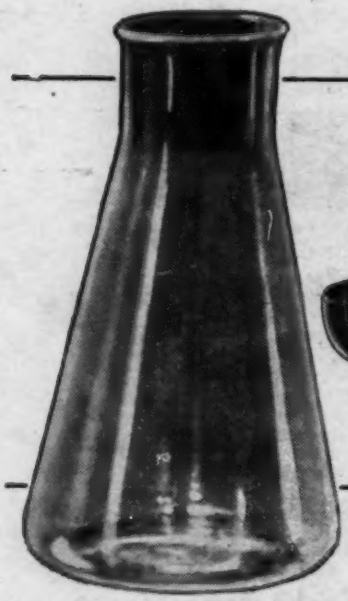

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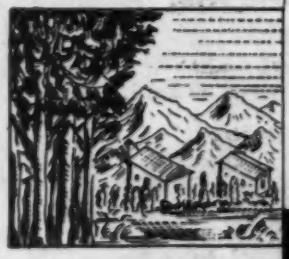

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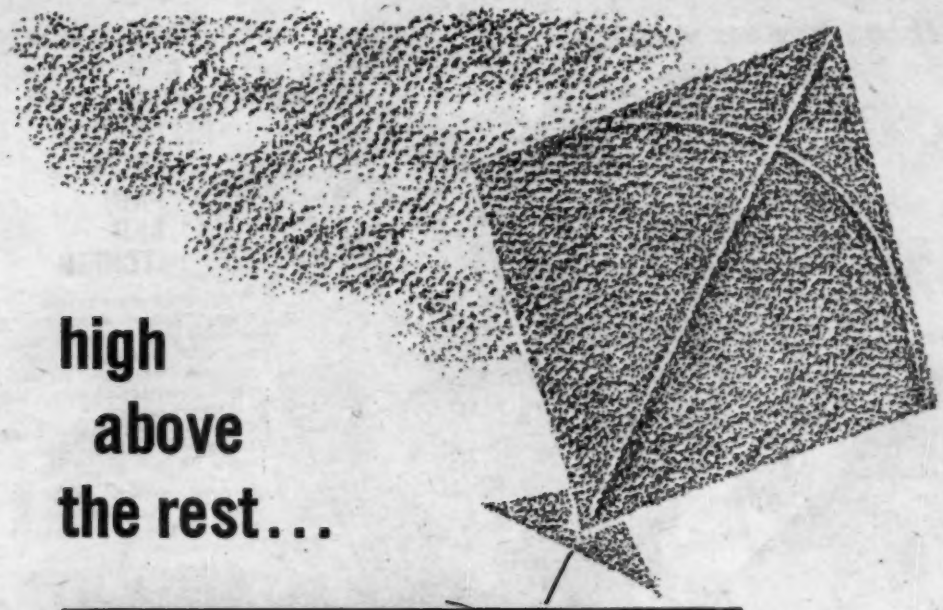

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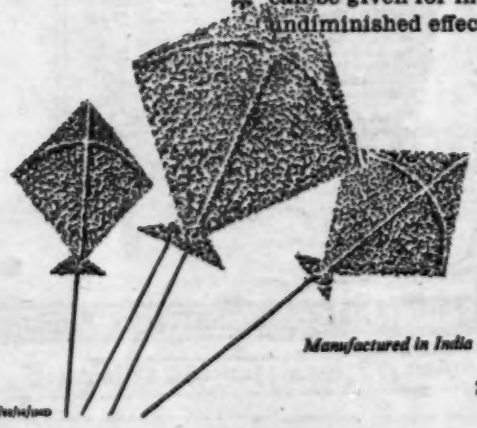

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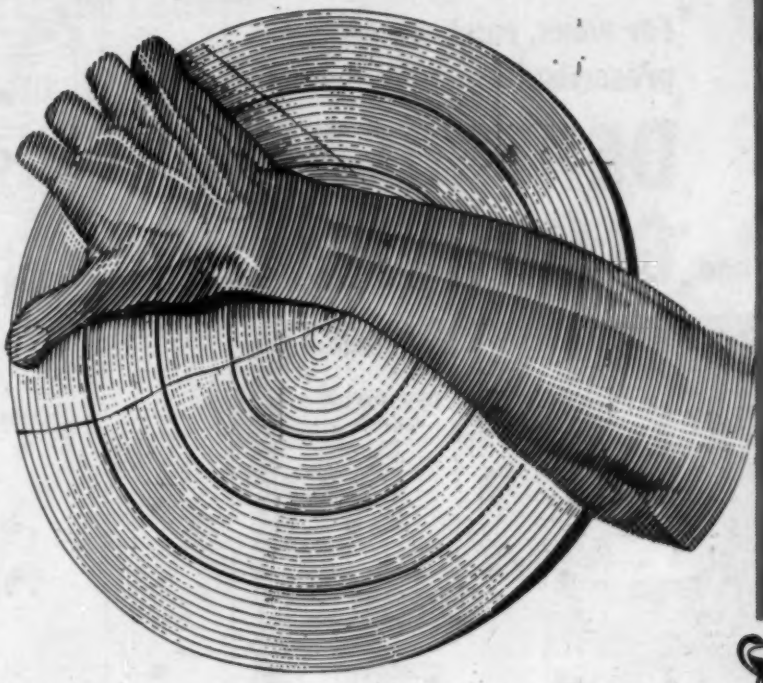

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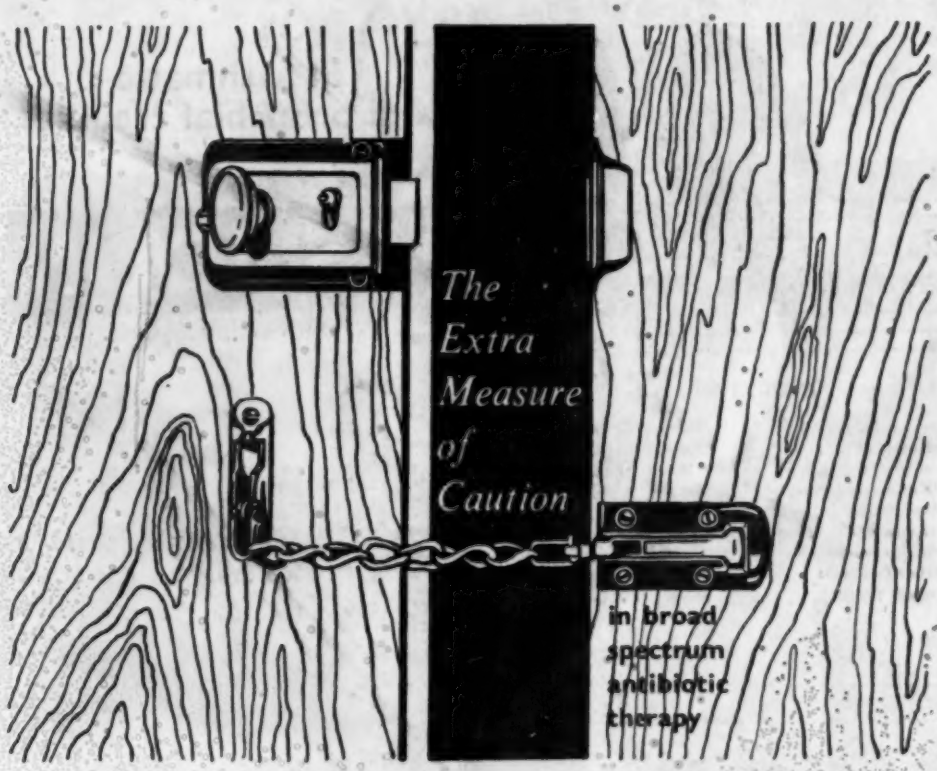

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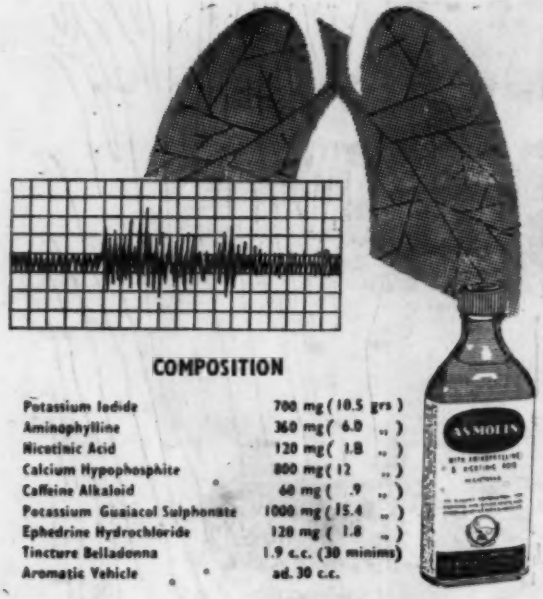

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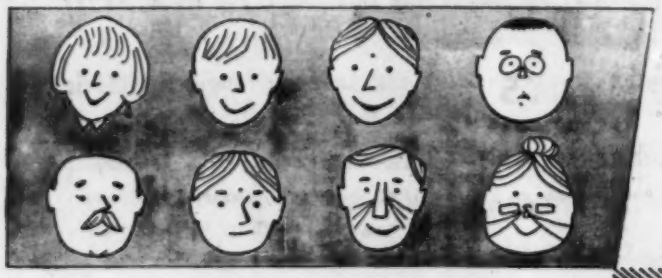

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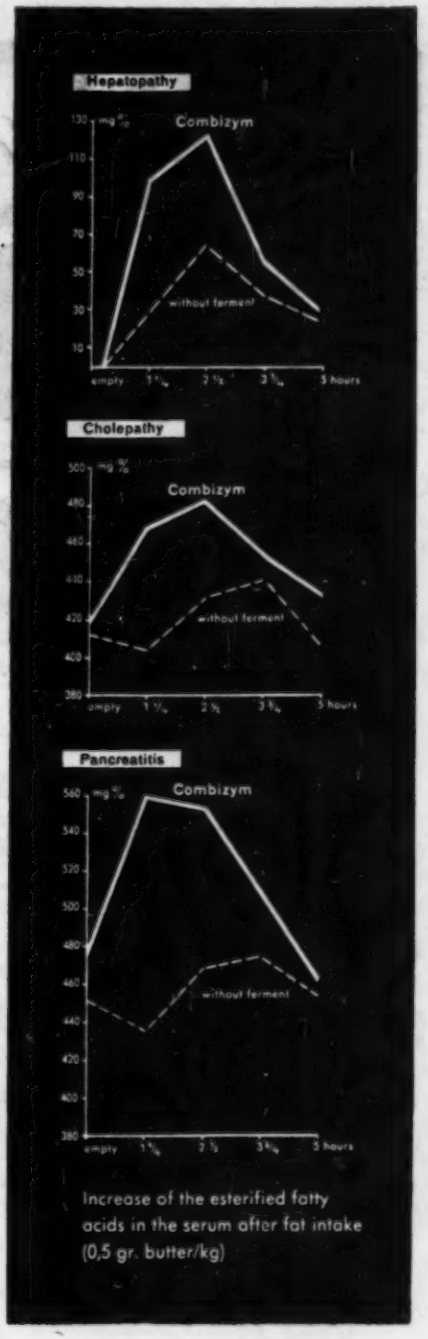

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ORIGINAL ARTICLES

SUBDURAL HAEMATOMA

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Subdural haematoma when diagnosed and treated in time forms one of the most gratifying experiences in neurosurgery. Ever since the description by Virchow the subject has been of great interest from many points of view.

Subdural haematoma is classified into acute, subacute and chronic varieties. The earlier descriptions in the literature were mainly about the chronic variety, and the inflammatory theory was upheld as the main aetiological factor. The first attempt at classification resulted in two varieties—acute and chronic.

The acute subdural haematoma usually complicates a head injury and often adds considerably to the difficulties in the management of such a case.

The chronic variety usually results from a head injury sustained months or years before the onset of symptoms.

The subacute variety comes somewhere in between. It usually results from an injury which has been sustained within six months. This is an arbitrary limit. The difference between the subacute and chronic variety is mainly in the age.

ANATOMY

A consideration of the anatomy of subdural haematoma requires a knowledge of the structure of the dura particularly with regard to its blood supply.

The dural vascular structure can be considered in three layers. The outermost layer consists of a wide mesh of capillaries, the next layer contains the main meningeal vessels, and the third or the innermost layer consists of an intricate, closely woven mesh of capillaries (Christensen, 1944).

The subdural space is a distinct space which contains the terminal portions of the veins that drain into the venous sinuses of the brain.

STRUCTURE OF THE HAEMATOMA

An acute subdural haematoma, when found without an associated laceration of the brain, consists of a collection of blood and clot in the subdural space.

But a non-acute one consists of an outer membrane and an inner membrane with the clot in between. The outer membrane when examined histologically consists of fibrillary connective tissue with plenty of vessels which run parallel to the inner layer of the dura. Actually one can find connections between the capillaries of the inner layer of the dura and those of the outer membrane. The inner membrane, on the other hand, consists of fine collagen and there are no blood vessels. Though

it lies on the arachnoid it can be very easily peeled off. The 'haematoma' in between consist of mushy and dark clots in various stages of liquefaction. Very occasionally the contents may be clear fluid, but then whether these can be classified in the same category as the one we are considering here is difficult to decide. The outer and inner membranes become continuous at the fornix. When this fornix is traced outwards it is found to merge with the inner layer of the dura.

These membranes are formed 2-3 weeks after the injury and with the passage of time they tend to become more fibrillary and less vascular.

Mechanism of Production

The subdural haematoma of traumatic variety only is considered.

The inflammatory process has been held responsible for subdural haematoma. While the role of inflammation cannot be denied, it must be remembered that often the injury that produces the haematoma is a minor one and is likely to be forgotten by the patient.

Acute subdural haematoma is usually a part of the picture of head injury and usually results from intrinsic injury to the brain. An underlying brain laceration is found in many of these cases. Sometimes acute subdural haematoma may result without an intrinsic injury to the brain, and here the mechanism is the same as in the chronic variety.

The mechanism of production of a subacute or chronic subdural haematoma is as follows: When the head receives a minor injury the relative movement of the various structures (i.e. skull, membrane and brain) depends upon their inertia and elasticity. As a result there is a lag between the response of the skull and the brain. So the slender veins that cross the subdural space get torn and bleed into the subdural space. Since most of the veins are found near the sagittal sinus it is no wonder that most of the haematomas are found over the convexity.

This explanation is quite simple and in its lucidity lies many weak points. The first objection is that if a minor head injury produces such a state of affairs why is not a subdural haematoma quite common. Actually subdural haematoma (in spite of its great importance) does not form more than 1 per cent of head injuries and minor head injuries are quite common. This objection has been circumvented by some, by supposing an individual predisposition resulting from fragility of the blood vessels. The second objection is that if the haematoma is the result of extravasation of blood why is it that it is always found in the convexity—actually the blood must gravitate to the base. But this again is not tenable since most of the veins are in the vault and again as the haematoma forms, it distorts the brain and pushes it all around, thus sealing the lower compartment.

If a subdural haematoma is due to a bleeding from the inner layer of the dura into the subdural space then how can one explain the formation of the inner membrane. Is it formed by the haematoma which organises or is it a reaction of the arachnoid? If it were due to the arachnoidal reaction then why is it not adherent to it? If it were the result of organisation of the haematoma then it is quite an unusual way of organisation of a haematoma, for a haematoma organises in its entire extent and not from the periphery inwards.

Again the experimental production of subdural haematoma requires two things: (a) injection of blood into the subdural space and (b) simultaneous ligation of the superior sagittal sinus.

Thus these objections do make us feel whether after all subdural haematoma can result from a primary intradural bleeding which spreads out the layers of the dura and forms for itself a cavity. This theory explains (a) the adherence of the outer membrane to the dura, for it is really part of the dura, (b) the absence of adherence between the inner membrane and the arachnoid, for this is the real subdural space, and (c) it also explains the formation of the fornix.

Signs and Symptoms

Subdural haematomas may present a variety of symptoms ranging from the minimal to the most serious and emergent.

Acute subdural haematoma is of two types (Whalley, 1949). One variety of acute subdural haematoma accompanies a gross injury to the brain. The symptoms and signs produced by this variety are merely grafted on to the picture of the brain damage. Possibly the mortality that results even after removal of the haematoma in these cases is due to the intrinsic injury to the brain. The second variety of acute subdural haematoma produces symptoms and signs similar to those of middle meningeal haemorrhages. Thus the classical picture of a lucid interval preceding increasing drowsiness may mean extradural or subdural haematoma. These cases give best results if operated in time.

The subacute variety of subdural haematoma can produce symptoms similar to those of many types of intracranial pathology. The suspicion of a subdural haematoma is raised if a history of head injury is asked for and is forthcoming. But oftentimes the head injury is a minor one and is

forgotten by the patient. The patient may be investigated for raised intracranial tension and a subdural haematoma found unexpectedly.

Case 1 (Case No. 11, vide Table 1) :

Patient K. was admitted for headache and vomiting of three months' duration. An inquiry into the history did not reveal any pertinent point. He also denied any history of head injury.

Clinical examination revealed a bilateral papilloedema and nothing else. An x-ray of the skull and an E.E.G. failed to reveal any abnormality.

In view of the complete absence of any positive neurological lesion, and as a preliminary step bilateral post burr holes were made under local anaesthesia. Both the ventricles were entered and were found to be under tension. But the surgeon felt that on the left side there was a sense of resistance.

On the basis of this single lateralising sign a left carotid angiogram was taken. This revealed the typical picture of a subdural haematoma.

The patient was operated upon. A big subdural haematoma was found over the left parietal cortex. During the operation massive cerebral oedema occurred which complicated the postoperative period for the first three days. The rest of the period was uneventful and the patient was discharged cured.

At times though there may be history of injury, the signs and symptoms may be very mild but quite enough to raise a suspicion of subdural haematoma. These cases can be confirmed by ancillary investigations.

Case 2 (Case No. 12, vide Table 1) :

Patient S. was admitted for headache particularly on the right side since two months. He was involved in a car accident 2 months ago but was not sure of any definite injury to his head. But since then he has had increasing headache and dimness of vision.

Clinical examination revealed papilloedema and there was slight spasticity on the left side with increased reflexes. E.E.G. was done and was found non-contributory. A right carotid angiogram revealed a typical subdural haematoma.

The patient was operated upon, and has attained good relief.

Sometimes the clinical picture is full of signs and symptoms which are definitely localisable to an important area of the brain. Further the drowsiness may come on suddenly and increase alarmingly.

Case 3 (Case No. 10, vide Table 1) :

Patient R was admitted with drowsiness and weakness on the left side of the body. The history was that two months ago he fell down and injured his chin. Two weeks later he found that he could not use the left upper limb properly and he used to drop any article he took in that hand. Gradually people around him found that there was an increasing unawareness of the left side of his body. His memory had been defective since 2 weeks and he was having a severe headache since last twenty days. His drowsiness was increasing gradually, and clinical examination revealed an early papilloedema, weak left abducens, a left hemiplegia and astereognosis.

An E.E.G. localised the lesion to the right temporal region but could not determine its nature.

A right carotid angiogram revealed a typical picture of subdural haematoma. This was evacuated by a right osteoplastic flap. His recovery was also very rapid.

An analysis of the cases that have been seen during the last few years is shown in Table 1.

TABLE 1—SHOWING ANALYSIS OF THE TWELVE CASES OPERATED

Case No.	Interval between injury & appearance of symptoms	Variety	Clinical signs	Investigated for only increased intracranial tension
1	One week	Subacute	Fits	—
2	Immediate	Acute	Coma	—
3	No history	Chronic	Secondary optic atrophy	Yes
4	One month	Subacute	Papilloedema	Yes
5	Five weeks	—do—	—do—	Yes
6	Eight weeks	—do—	Left hemiparesis, attacks of giddiness	—
7	Six weeks	—do—	VI N. palsy, papilloedema	Yes
8	Few hours	Acute	Left hemiparesis	—
9	Unconscious condition	—do—	—	—
10	Eight weeks	Subacute	Parietal lobe syndrome	—
11	No history of injury	—do—	Papilloedema	Yes
12	Eight weeks	—do—	Slight spasticity of left side	—

All the cases were cured except Case No. 2 which expired.

DIAGNOSTIC PROCEDURES

A plain x-ray of the skull may not reveal much except in the occasional case of a calcification in a subdural haematoma.

By far the most important investigation in a suspected case of subdural haematoma is carotid angiography. Carotid angiography is sufficient in most cases, as the majority of subdural haematomas are over the frontal, temporal or parietal regions. In the anteroposterior view the pictures

reveal an avascular area separating the vesels from the inner table (Figs. 1 and 2, *vide* Plate). The lateral view shows the displacement of vessels which may indicate the longitudinal extent of the tumour (Fig. 3, *vide* Plate).

Provided there is no contra-indication air studies may confirm or raise a suspicion of subdural haematoma.

Electroencephalography perhaps constitutes the second valuable test in these cases.

Treatment

The treatment of a subdural haematoma depends on its age.

The acute subdural haematoma which complicates a parenchymatous injury to the brain perhaps needs no special treatment—as indicated before, it may seldom be recognised for that matter. Again unless definite signs of compression are present the results of operation are not encouraging.

The second variety of acute subdural haematoma is indistinguishable from extradural haematoma. If in a suspected case of extradural haematoma no haemorrhage is found on the dura, then the subdural space must be tapped (if necessary a small nick may be made) and the presence or absence of a subdural haematoma verified. If cerebrospinal fluid escapes, then obviously there is no subdural haematoma. If a subdural haematoma is found, the dural opening is enlarged and the haematoma sucked out. This may be sufficient in most cases.

A subacute or chronic subdural haematoma needs a more elaborate procedure. The signs and symptoms in these cases are due to the expanding nature of the haematoma and the inner membrane which incarcerates the brain. The treatment of a chronic subdural haematoma involves an osteoplastic craniotomy. When the dura is exposed the discoloration of the dura confirms the diagnosis. The dura is then opened and the haematoma which consists of varying amount of solid clots and sanious fluid is removed. The outer membrane is then removed. Then the inner membrane is peeled away from the underlying pia. The plane of cleavage is distinct and separation is quite easy. Care must be taken at the various sulci to prevent injury to the pia and the vessels. As the membrane is traced all round the fornix is reached. The fornix can be left *in situ* if its removal may mean extra dissection or extension of the flap.

After the removal of the inner membrane time must be allowed for the brain to expand. This is very necessary since failure of expansion of the brain may result in fresh bleeding and recurrence of symptoms. The expansion of the brain must be complete by about 5-6 minutes. If it does not do so then the aid of the anaesthetist should be sought. The addition of a little CO_2 to the anaesthetic mixture may help. If this fails lumbar puncture and injection of 20 ml. of sterile distilled water can be tried. In some cases intraventricular injection of sterile saline can be given. In Case 10 (Table 1) we had to inject about 25 ml. of sterile saline after a trial of the other measures and before a satisfactory expansion of the brain could be achieved. Occasionally, if despite all these measures the brain does not expand a polythene tubing can be led out from this space and the wound closed. Subsequently gentle suction is applied to this tube.

Ironically enough in some cases after the removal of the inner membrane the brain might become oedematous and bulge in a most annoying manner familiar to all neurosurgeons. The cause of this oedema is perhaps similar to that which occurs in other operations on the brain.

In the series under review, in Case 12 (Table 1) the oedema was so much that the dura could not be closed. Hence gel-film had to be used.

Results

An analysis of the 12 cases operated on by the authors is given in Table 1. As has been remarked at the beginning of the article, properly timed operations for subdural haematoma form one of the most gratifying chapter in neurosurgery.

The recovery in an acute case is quite quick (as is common in all cases of posttraumatic compression of the brain).

In chronic cases the recovery is quite fast when compared with recovery from tumours, etc. In Case 10 (Table 1) where there was a complete parietal lobe syndrome, the patient was aware of the affected side within 48 hours and at the time of discharge (i.e., about 15 days from date of admission) he had almost completely recovered.

Summary

The pathogenesis and clinical symptoms of subdural haematoma are discussed. Some cases are presented in detail to illustrate the clinical picture. An analysis of the twelve cases operated upon is also presented.

Acknowledgment

Our thanks are due to the Dean, Government General Hospital, Madras, for permission to publish this article.

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Figs. 1 and 2—Roentgenograms of the skull (anteroposterior view) showing characteristic avascular area (shown by arrows) separating the brain from the inner table of the skull.

Fig. 3—Roentgenogram of the skull (lateral view) showing the looping and displacement of the vessels caused by the haematoma.

BALASUBRAMANIAN AND RAMAMURTHI—*Subdural Haematoma (p. 1)*

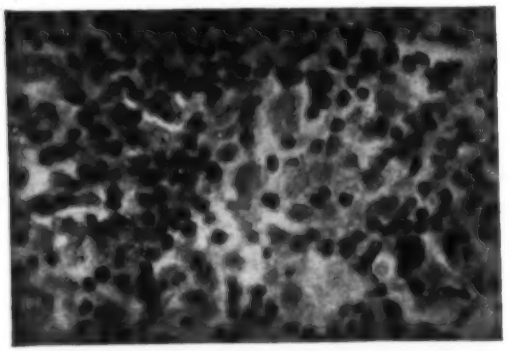

Fig. 1—Section of lymph gland showing parasitised histiocytes surrounded by round cell infiltration (H. & E., low power).

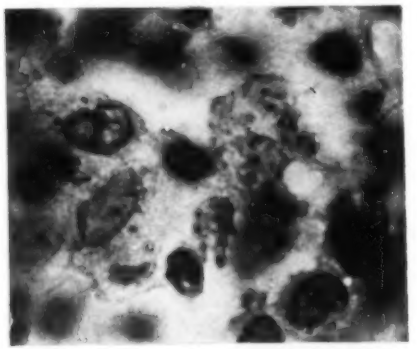

Fig. 2—Showing parasitised histiocytes from section in Fig. 1 (under oil immersion).

SEN GUPTA AND CHATTERJI—*Lymphadenopathy in a Case of Indian Kala-azar (p. 21)*

Fig. 1—Skiagram of the left wrist showing localised cystic lesion in the carpus.

Fig. 2—Skiagram of left shoulder showing a few small discrete radiolucent cystic lesions in the upper end of the right humerus.

BOSE—*Multiple Pseudocystic Tuberculosis of Bone in an Adult (p. 22)*

Fig. 1—Skiagram of the right femur showing fracture of middle of the shaft with malunion.

Fig. 2—Skiagram of the left femur showing fracture of middle of the shaft.

Fig. 3—Skiagrams of both tibiae and fibulae showing fractures.

JAIN AND SANGHVI—*Osteogenesis Imperfecta Tarda (p. 24)*

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CIRRHOSIS OF THE LIVER WITH SPECIAL REFERENCE TO INTRAVENOUS LIVER EXTRACT THERAPY

A Study of 35 Cases

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Cirrhosis of the liver has always presented a challenge to the medical profession since the time of Eristratus who first described it in 300 B.C. and Laennec who wrote its first adequate account in 1819. It is unfortunate that there is no satisfactory treatment of such a chronic and progressive illness. Many treatments have been tried over years in the past by workers like Beams (1946), Fleming and Snell (1942), Labby *et al* (1947), Patek *et al* (1948) and Rimmerman *et al* (1944) with controversial results. Studies on cirrhosis of the liver in human beings have served to emphasise the importance of the nutritional state. Dietary regimens have been suggested by various workers like Fleming and Snell (*loc. cit*), Patek *et al* (*loc. cit.*) and Rimmerman *et al* (*loc. cit*), with a protein quota of 100-140 g., and caloric value of 3000-3500 calories, and a generous vitamin supplement and powdered yeast. Beams (*loc. cit.*) and Cayer and Cornatza (1949) reported the beneficial effects of choline in treating patients with cirrhosis of the liver. Parenteral protein digests and aminoacids (Lewis *et al*, 1947) did not show consistently demonstrable therapeutic results. Post *et al* (1951) showed that human albumin or plasma may be a useful adjunct in the management of a severely decompensated cirrhosis. However, the unavoidable high cost and the hazards of its administration render this form of treatment impracticable for routine use.

Liver extract or liver substances have been extensively used by oral, intramuscular and intravenous routes by Aldercruetz and Gustaf (1953), Drill and Hall (1950), Labby *et al* (*loc. cit.*), Ley *et al* (1955) and Rimmerman *et al* (1944) with beneficial results. Liver extract has been extensively used parenterally and it has been customary to give it by the intramuscular route. Crude liver extract, used intravenously in large doses, has proved to be of value. A study was initiated by Labby *et al* (*loc. cit*) and encouraging results were reported. The work was further carried out by Ralli *et al* (1949) who studied the cases in two groups of patients by giving them the liver extract either intramuscularly or intravenously. The results were better in the latter group. The rationale of using a crude liver extract by vein has been suggested because in a long standing case of cirrhosis of the liver there exists a muscular wasting which reduces the rate of absorption and thus increases the tendency to produce a sterile abscess (Labby *et al*) after intramuscular injection. Moreover when in such a wasted muscle 10-15 c.c. injections are introduced it becomes so painful that the patient refuses further intramuscular therapy. Thus recently workers like Aldercruetz and Gustaf (*loc. cit.*), Drill and Hall (*loc. cit.*), Kalk and Wildhirt (1953) and Sarin and Mittal (1955) from different centres have tried intravenous therapy and reported beneficial results.

This paper describes the plan of using crude liver extract by intravenous route and our observations which indicate its possible therapeutic effectiveness.

Material and Methods

The material consisted of 35 patients with cirrhosis of the liver admitted in the medical wards of M. Y. Hospital, Indore. The clinical history was noted in every case with special reference to gastrointestinal symptoms and diet. A thorough general examination was made with particular attention to skin, icterus, and oedema. Apart from the routine general and systemic examinations the size of the liver and the spleen, the presence of abdominal veins and ascites were noted with particular care. The diagnosis was aided by haematological examination, roentgenological examination, including percutaneous splenoportography, and the liver function was assessed by a wide spectrum of tests. Confirmation of diagnosis was done by needle biopsy of the liver.

The patients were in a decompensated stage of cirrhosis as indicated by icterus, oedema and ascites. No planned criterion was exercised in selecting the patients beyond that each case was manifestly decompensated but not terminal cirrhosis. The patients were hospitalised and kept there for a maximum period of time. When compen-

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sated the patients were allowed to take outdoor treatment and could come from their home for regular check-up.

The patients were kept on the hospital vegetarian diet which supplied roughly 1800 calories. Curd or milk (half a seer) was given to all the patients to make up for the deficiency of protein in the diet. No lipotropic substance was administered.

Plan of Therapy with Crude Liver Extract

Crude liver extract was administered in all the cases after testing the sensitivity. Sensitivity was tested by injecting 0·2 c.c. of undiluted liver extract intramuscularly. The condition of the patient was watched for any untoward reactions such as flushing of the skin, urticaria, headache, difficulty in breathing, cough, etc. If there was no reaction 0·5 c.c. of liver extract diluted to 25 c.c. with sterile solution of 5 or 25 per cent glucose was given very slowly intravenously.

If the patient did not have any reaction, the following scheme of dosage was employed:

First day	...	1 c.c.	diluted to	25 c.c.
Second day	...	2 c.c.	diluted to	50 c.c.
Third day	...	3 c.c.	diluted to	50 c.c.
Fourth day	...	5 c.c.	diluted to	50 c.c.
Fifth day	...	7 c.c.	diluted to	50 c.c.
Sixth day	...	10 c.c.	diluted to	50 c.c.
Seventh day	...	12 c.c.	diluted to	50 c.c.
Eighth day	...	15 c.c.	diluted to	50 c.c.
Ninth day	...	20 c.c.	diluted to	50 c.c.

The maximum dose given was 20 c.c. diluted to 50 c.c. After about 10 daily injections of 20 c.c. each the drug was administered less frequently—first on alternate days, then biweekly, weekly and at fortnightly intervals. The average period of follow-up was 6 months.

Clinical Observations

Age and sex—The details of the distribution of age and sex are given in Table 1.

Table 1—Showing Age and Sex Distribution of the Patients

Age (years)	Males	Females	Number	Percentage
15-20	2	1	3	8·56
21-30	3	4	7	20·00
31-40	5	7	12	34·30
41-50	9	1	10	28·50
51-60	3	—	3	8·56

There were 22 males and 13 females and 82·8 per cent of the cases belonged to the age-group 21-50 years. All the patients belonged to the poor-income group.

An analysis of the presenting complaints is given in Table 2.

Table 2—Showing Analysis of the Presenting Symptoms in Relation to Incidence

Presenting complaints	No. of cases
Distension of abdomen	35
Fatigue	35
Loss of weight	35
Anorexia	34
Flatulence	34
Oliguria	33
Oedema	26
Diarrhoea	20
Pain in abdomen	19
Jaundice	4
Haematemesis	3

General complaints of a feeling of tiredness, and loss of weight were present in all the patients. Digestive disorders like anorexia, distension of abdomen, a discomfort after meals, flatulence and irregular bowels were present in over 90 per cent of the patients. Only 4 patients complained of jaundice though it was manifest in 7. Three cases presented with haematemesis.

Diet—The duration of complaints varied a great deal, from 1 month to over 3 years, with an average of six months. The dietetic data showed that the diets of 85 per cent of patients were unbalanced and consisted of rice or *atta* which lacked in protein. Even though 34·3 per cent of patients were non-vegetarians none of them could afford to take either egg, meat or fish more than once a week.

Alcoholism—More than 50 per cent of the patients were addicted to alcohol and 81·2 per cent of these had been consuming 8-12 chhataks of alcohol per week for a period of 10 year or over.

Past illness—History of jaundice could be elicited in 42·9 per cent cases and 33 per cent of these had never felt well after the attack of jaundice, leading them eventually to the present state of health. A history of chronic diarrhoea was available in 31·35 per cent of cases. The history of periodic fever with or without chill was present in 57·14 per cent.

All the patients looked pale and emaciated and obvious muscular wasting was present in 75 per cent patients. Signs like spider naevi, palmar

erythema, and testicular atrophy were rare findings. Spider naevi were seen only in one patient who was a Chinese national. Gynaecomastia was also observed in one case only. Details of the physical signs have been given in Table 3.

TABLE 3—SHOWING ANALYSIS OF CLINICAL FEATURES IN RELATION TO INCIDENCE

Clinical features	No. of cases
Ascites	35
Splenomegaly	34
Collateral circulation	33
Anaemia	26
Oedema	26
Icterus	7
Clinical avitaminosis	7
Oesophageal varices	6
Clubbing of fingers	2

In 75 per cent of cases, a paracentesis abdominis had to be performed. In certain instances this was undertaken even before the treatment was instituted, for relieving the distress of the patient.

The spleen was palpable in 97 per cent of the cases, and in 8·56 per cent it was disproportionately enlarged. In all of them a history of malaria was forthcoming. Visible veins over the abdomen could be seen in 92 per cent of the patients. The liver was found enlarged enough to be felt below the costal margin in 57·14 per cent of cases. It was not palpable in 34·3 per cent cases and in another 11·4 per cent cases only the edge of the left lobe could be first palpated at the xiphisternum indicating that the liver was retracted in these patients.

LABORATORY FEATURES

Haematological investigations showed that 80 per cent of the patients had haemoglobin 8 g. per cent or over and the maximum number of patients (60 per cent) had R.B.C. count ranging from 3-4 mill./c.mm. ; none had R.B.C. count below 2 mill./c.mm.

Urine and stool—Routine examinations showed the presence of bile salts and pigments in 6 patients and cystic forms of *Entamoeba histolytica* in 2 cases.

Ascitic fluid—Examination of ascitic fluid revealed that the proteins were below 1,000 mg. per cent in 32·9 per cent of cases. Out of 6 patients who had proteins over 2,000 mg. per cent, 3 gave evidence of tuberculous infection in the lungs. Barium meal examination of the oesophagus for varices was done in all the cases. The presence of varices could be demonstrated in only 5 patients.

In the absence of any single reliable test for the functional capacity of the liver, a battery of liver function tests was employed for assessing the degree of the derangement. These included measurement of total plasma proteins with determination of albumin : globulin ratio, prothrombin time, icteric index, van den Bergh's test, cephalin-cholesterol flocculation, thymol turbidity and flocculation tests and urine urobolinogen. Out of these liver function tests serum albumin-globulin estimations, thymol turbidity and cephalin cholesterol flocculation tests were found to give abnormal results in the maximum number of cases.

Adopting the Indian standard as worked out by Dutta and Chakravarty, a hypoalbuminaemia was observed in 91·5 per cent of the patients. Hyperglobulinaemia was also found in a similar number of patients leading to the reversal of albumin : globulin ratio.

Splenoportography was performed in 19 patients. The results obtained by the procedure are shown in Table 4.

TABLE 4—SHOWING THE SPLENOPORTOGRAPHIC PATTERNS

Splenoportography pattern	No. of cases	Percentage
Tortuosity of splenic and portal veins	8	40·1
Oesophageal varices	5	26·2
Blocked portal vein and varices	1	5·3
Double splenic vein	1	5·3
Dye in splenic capsule ...	3	15·8
Spilling of the dye in the peritoneal cavity	1	5·3

Varices were present in 5 patients and in 4 a history of haematemesis was present. In one patient the splenic and portal veins were not seen and there was a bunch of collaterals, suggesting a blocked portal vein with anastomotic channels. On 3 occasions the patterns obtained were either a collection of the dye in the splenic capsule or did not furnish any information. All the 5 patients who had varies were in an advanced stage of the disease. The spleen was found 2-4 fingers enlarged. Liver was retracted in 4 of them. The average duration of decompensation had been 10 months and the liver functions were either severely or moderately deranged.

Histopathological observations—Liver biopsy was attempted in all the patients. The liver

biopsy material was studied in 25 cases and their histological patterns have been summarised in Table 5.

TABLE 5—SHOWING HISTOLOGICAL PATTERNS OF 25 CASES

Histological patterns	No. of cases	Percentage
Established cirrhosis ...	9	36·00
Postnecrotic scarring with nodular hyperplasia	6	24·00
Early cirrhosis	3	12·00
Fatty infiltration of liver ...	3	12·00
Swelling of liver parenchyma ...	3	12·00
Biliary cirrhosis	1	4·00

A large number of specimens showed the presence of established cirrhosis, 9 showed diffuse hepatic fibrosis, 6 post-necrotic scarring with nodular hyperplasia and one biliary cirrhosis. Only 12 per cent showed early cirrhosis. In 3 there was a fatty infiltration and a similar number showed only swelling of liver parenchyma and cellular infiltration. Where advanced changes were encountered, the liver functions were found more severely deranged.

RESULTS OF I.V. LIVER EXTRACT THERAPY

Liver extract was administered by intravenous route, according to the schedule mentioned above, to 35 patients. In 3 patients the investigations could not be completed and in another 6 the therapy had to be abandoned for untoward reactions. The duration of follow-up in the remaining 26 cases is summarised in Table 6. It will be seen

TABLE 6—SHOWING THE MONTHS OF FOLLOW-UP

Period of follow-up (months)	No. of cases	Percentage
1—3	6	22·7
3—6	13	50·0
6—9	2	7·5
9—12	4	15·1
Over 12	1	3·7

that 6 patients had the treatment for less than 3 months. This number was considered too small for the assessment of therapy.

The various untoward reactions encountered in these patients are summarised in Table 7. Minor reactions like heaviness of head, headache, palpitation, flushing of skin, etc., were commonly met with, and in most cases they could be avoided if the rate of administration of the drug was slowed down. The itching and urticaria met with in two patients responded to antihistaminics. One case developed pulmonary oedema and could not be saved. Only on 6 occasions were the reactions severe enough to necessitate discontinuation of the drug.

TABLE 7—SHOWING THE UNTOWARD REACTIONS ENCOUNTERED

Untoward symptoms	Percentage
Heaviness in the head	50·00
Throbbing and headache	37·50
Palpitation	25·00
Itching and urticaria	6·25
Pulmonary oedema	3·13

Clinical progress—The clinical improvements noticed in patients who were on intravenous liver extract therapy for more than 3 months are summarised (Table 8). One of the first symptoms of improvement was a return of appetite which was noticed in 95 per cent of cases. Of these in 25 per cent the appetite became hearty where it was practically non-existent before the commencement of the therapy. The feeling of well-being and diminution in fatigability almost coincided with the improvement in appetite. The tissue wasting was checked and the patients showed a steady gain in weight. The appearance of flesh in previously wasted areas gave sufficient evidence of this fact. Most of the patients became more active than before.

TABLE 8—SHOWING THE CLINICAL IMPROVEMENT IN PATIENTS AFTER THE THERAPY

Symptoms	No. of cases	Percentage
Feeling of well-being	19	95·00
Improvement of appetite ...	19	95·00
Gain in weight	18	90·00
Improvement of anaemia ...	15	75·00
Increase in urine output ...	15	75·00
Decreased frequency of paracentesis	14	70·00
Disappearance of ascites ...	4	20·00

The most satisfactory sign of improvement was the retardation in the accumulation of fluid in the peritoneal cavity. Most of the patients had been

subjected to paracentesis before the therapy was started. The number and frequency of paracentesis were remarkably reduced in 14 patients and in another four (20 per cent) the ascites completely disappeared after 3-12 months of treatment.

Occasional patients showed a moderate periodic diuresis after an intravenous injection of liver extract and all the patients showed gradual increase in urine output.

The clinical improvement was accompanied by a simultaneous improvement in the serum protein levels and the tests of hepatic function.

Tables 9 and 10 show the biochemical improvement after treatment with liver extract.

TABLE 9—SHOWING BIOCHEMICAL IMPROVEMENT AFTER THE TREATMENT

Serum proteins	Percentage of patients	
	Before treatment	After treatment
Total proteins, 6 g. per cent or higher	65	95
Serum albumin, 3·5 g. per cent or higher	20	70
Serum globulin, 2·5 g. per cent or higher	90	60
A :G ratio, 1·5 or higher ...	10	45

TABLE 10—SHOWING THE DEGREE OF LIVER FUNCTION DERANGEMENT AFTER THE TREATMENT

Degree of derangement	Percentage of patients	
	Before treatment	After treatment
Mild	10	30
Moderate	30	45
Severe	60	25

The gradation of liver function derangement as mild, moderate and severe damage has been assessed from the average of all the liver function tests.

Before the commencement of the therapy only 20 per cent patients had serum albumin level of 3·5 g. per cent or over, while after the treatment such levels were found in 70 per cent of patients. Serum globulin level showed a decline in 30 per cent of patients. This brought the albumin : globulin ratio to 1·5 or higher in 45 per cent of patients from the pre-treatment number of 10 per cent.

The incidence of gross liver derangement fell from 60 to 25 per cent and mild derangement was encountered in 30 per cent of the patients in contrast to the pre-treatment figure of 10 per cent.

An idea about the improvement of cases on the therapy can be obtained from the following selected cases of the series:

CASE REPORTS

Case 12—B. K., aged 35, male, tailor, complained of gastrointestinal symptoms for the last 6 years after a presumable attack of infective hepatitis. He had complete loss of appetite and gradually increasing abdominal distension. On admission he looked pale and emaciated. His spleen was 7 fingers enlarged below the costal margin; and the liver was retracted with a palpable left lobe. Barium meal examination and splenoportography successfully revealed oesophageal varices. The liver function tests showed moderate derangement and the histopathology an early cirrhosis. With I.V. liver extract the appetite improved, oedema subsided and the urine output increased within a month of therapy. Ascites completely disappeared in 5 months. Liver functions also showed improvement. The patient is still under observation.

Case 18—S. R., aged 35, male, labourer, complained of anorexia, flatulence and ascites of 3 months' duration. He had been a chronic alcohol addict. His liver was 2 fingers enlarged, the spleen was palpable and ascites was pronounced. The liver function tests showed moderate derangement and the histopathology showed established cirrhotic pattern. The full schedule of intravenous liver extract therapy was instituted. The general symptomatic improvement was evident after a month, and the need for a paracentesis was felt 3 months later. Thereafter the urine output increased and the ascites formation was markedly retarded. Liver functions showed a recovery. The patient is still being followed up. He receives 20 c.c. of liver extract intravenously every fortnight.

Out of the 35 patients in the series there were 6 deaths. All of them had come in a fairly decompensated state. 3 of them died in hepatic coma, and one during an exacerbation of respiratory infection. The latter developed sensitivity to liver extract, got pulmonary oedema and could not be revived. In the other two, death was precipitated by haematemesis.

Out of the total number of 20 patients, 4 became completely asymptomatic and are now on treatment-free observation. Another 13 patients showed considerable improvement. In the remaining three cases the course of the disease appeared to be unaltered.

Comments

The assessment of a therapeutic regime in chronic illness is rendered difficult by several factors. All the cases recorded here stayed in the hospital for a considerable period and the minimum period of follow-up which was considered to have brought forth a material change was 3 months. Due to this long period of follow-up, a study with a greater number of patients was not possible.

Follow-up studies in the outpatient department were not possible as the patients usually came from distant villages, belonged to poor economic class, were less intelligent and could not be relied upon to follow the medical instructions at home. Their treatment as hospital inpatients afforded an opportunity for more regular medical supervision.

Digestive disturbances were the commonest finding in the series and anorexia was a most pronounced feature. The patients were encouraged to eat as much as they could. Any further deleterious effect of alcohol was avoided as the patients completely abstained from alcohol during their stay in the hospital.

Clinical vitamin deficiencies were treated with adequate doses of appropriate vitamins. When the patients regained their appetite after treatment with I.V. crude liver extract they could eat adequately and derive essential food factors from the natural sources. Rest and freedom from undue physical strain are as essential as the diet.

Histopathological studies are considered important in the study of cirrhosis, as they give a clue to the prognosis of these patients. Thus out of 6 patients who showed a postnecrotic scarring with nodular hyperplasia 4 succumbed within a period of 4 weeks' follow-up and the other 2 failed to show any improvement. Out of 9 patients with diffuse hepatic fibrosis 6 showed considerable clinical and biochemical improvement and the rest showed only symptomatic improvement. These findings are interesting and need further study in a larger number of cases before any definite conclusion could be drawn. In our series, all the patients who showed early cirrhotic changes or fatty infiltration with swelling of liver parenchyma, showed complete rehabilitation, comparing favourably with a state of apparently normal health.

The way in which intravenous liver extract helps has long been a subject of dispute. The substitution hypothesis fails to explain the results satisfactorily. So little is known about the chemical nature of crude liver extract that it has not been possible to indicate the constituent factors that might have met the requirements of this complex disease. Pteroyl glutamic acid (folic acid) and vitamin B_{12} in addition to various autogenous liver cell enzyme systems may play an important role (Kalk and Wildhart, 1953).

Drill and Hall (1950) demonstrated the lipotropic properties of liver extract. It is not unlikely that liver extract might encourage the regeneration of hepatic tissue and assist in restoring to normal the aberrations of water balance, enzyme synthesis, and energy metabolism that are known to occur in cirrhotic patients.

Ralli *et al* (1949) stated that crude liver extract contained many growth promoting factors that not only help tissue regeneration but also play some part by improving the appetite and reducing the amount of antidiuretic factors in the body.

The role of liver extract can be ascertained most easily by the return of appetite which in turn having a desirable effect on the storage of protein and the general nutritional state, tends to reverse the factors active in the production of hepatic decompensation.

Whether the observed therapeutic utility of the drug can permit us to postulate a deficient fraction, perhaps contained in the liver extract, time alone can tell.

Summary

Thirty-five patients of decompensated cirrhosis admitted into the medical wards of M. Y. Hospital, Indore, were studied for the therapeutic effects of intravenously administered crude liver extract, observed for a period of 6 to 18 months.

All the patients had long-standing gastrointestinal symptoms and came to the hospital when ascites had appeared. Their diets were grossly lacking in proteins of good biological value, and alcoholism was an associated feature in 50 per cent of patients. A history of presumable attack of infective hepatitis was available in 42·9 per cent of cases.

Biochemical investigations were carried out in the beginning of the study and the tests were serially performed, besides following up of the clinical progress of these patients. Histopathological studies were undertaken in most of the patients.

Percutaneous splenoportography yielded valuable information regarding the collaterals and the state of the portal circulation.

All the patients were kept on hospital diet supplemented with milk or curd. Twenty patients followed up for a period of 3 months and over showed symptomatic improvement manifested by a return of appetite, and return of vigour. The urine output gradually increased, oedema disappeared, and accumulation of ascitic fluid was materially reduced. Sixteen patients showed

considerable improvement, and 4 became completely asymptomatic.

It is felt that liver extract administered intravenously in large doses restores and sustains appetite. When used in conjunction with a full diet in properly selected cases, the drug may prove to be valuable in the management of cirrhosis of liver and if given an intensive trial, particularly in early cases, may prove successful in halting the pathological process.

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PROGNOSTIC FACTORS IN TUBERCULOUS MENINGITIS

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The advent of chemotherapeutic agents effective against the tubercle bacilli has now turned the agelong fight of mankind against tuberculosis in our favour. Today the judicious use of chemotherapy in suitable cases may mean complete recovery from the disease in almost all the cases. However, a hundred per cent survival and recovery rate is a highly elusive figure. In order to predict the ultimate course of the disease, a number of factors have been studied. While much work has been done in the past on these factors, their significance has varied with different observers. The present work was undertaken to find out such simple prognostic factors which could be practically worked out in any laboratory.

Material and Methods

55 cases of tuberculous meningitis admitted to the medical wards of Gandhi Memorial and Associated Hospitals and Kasturba T. B. Hospital, Lucknow, have been studied in detail. Out of these, 15 patients were treated entirely on chemotherapy which consisted of 1 g. of streptomycin given intramuscularly daily for 60 days and isoniazid in dosage of 10 mg./kg. body weight orally per day. After 60 days of therapy 8 g. of PAS orally per day was given in place of streptomycin, while isoniazid was continued as before. The remaining 40 patients were given 20 mg. of prednisolone per day orally in four divided doses in addition to the chemotherapy mentioned above.

Prior to initiation of the therapy, these cases were studied in detail. Clinical examination of the patients was conducted and their mental condition was classified according to the criteria of McCarthy and Mann (1950). Meningitis was classified into 3 stages according to the criteria of M.R.C. (1948).

Besides these 55 cases, 25 additional cases were selected from the old records of the hospital. All the cases of tuberculous meningitis admitted to the hospital during the last 4 years and treated with intrathecal streptomycin in addition to the usual chemotherapy were sorted out. Out of these, 25 cases were selected.

The laboratory studies included cellular and biochemical examination of C.S.F. Besides these

examinations, tuberculin test with 5 TU of PPD was conducted on each case every 15th day.

The clinical condition of the patients at the time of their discharge from the hospital was classified as having recovered or partly recovered according to the criteria of Misra and Khanna (unpublished).

Results

The final results obtained in the series under review have been analysed in relation to various factors. An analysis of these factors is presented in Tables 1 to 13 in each of which figures in parentheses indicate percentage of cases and I/T stands for intrathecal.

Table 1—Showing the Influence of Age on the Results Obtained

Age group (years)	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
15—20	4 (66)	1 (17)	1 (17)	1 (75)	1 (12·5)	1 (12·5)	11 (92)	1 (8)	—
21—25	—	1 (33)	2 (67)	—	1 (25)	3 (75)	4 (66)	—	2 (34)
26—30	1 (50)	—	1 (50)	4 (56)	1 (16)	2 (28)	7 (77)	1 (11·5)	1 (11·5)
31—35	—	—	1 (100)	—	1 (33)	2 (67)	5 (62·5)	1 (12·5)	2 (25)
36 & over	2 (66)	—	1 (34)	—	—	3 (100)	4 (50)	3 (37·5)	1 (12·5)

Table 2—Showing the Influence of Sex on the Results Obtained

Sex	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Male	1 (17)	1 (17)	4 (66)	3 (33)	2 (23)	4 (44)	6 (54)	2 (19)	3 (27)
Female	6 (66)	1 (12)	2 (22)	7 (44)	2 (12)	7 (44)	22 (75)	4 (14)	3 (11)

Table 3—Showing the Influence of the Stage of Disease on the Results Obtained

Stage of disease	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Intermediate ...	6 (100)	—	—	8 (73)	3 (27)	—	11 (69)	3 (19)	3 (12)
Advanced	1 (11)	2 (23)	6 (66)	2 (14)	1 (7)	11 (79)	17 (70)	3 (12)	4 (18)

N.B.—There was no case in the 'Early' stage of disease.

TABLE 4—SHOWING THE INFLUENCE OF MENTAL STATUS ON THE RESULTS OBTAINED

Mental status	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
II	3	—	—	8	—	—	—	—	—
	(100)	—	—	(100)					
III (a)	3	—	—	1	2	1	16	—	—
	(100)			(25)	(50)	(25)	(100)		
III (b)	1	2	3	1	2	6	12	5	1
	(17)	(33)	(50)	(11)	(22)	(67)	(66)	(28)	(6)
IV	—	—	3	—	—	4	—	1	5
			(100)			(100)		(17)	(83)

N.B.—There was no case in Stage I of mental condition.

TABLE 5—SHOWING THE INFLUENCE OF EXTRAMENINGEAL TUBERCULOSIS ON THE RESULTS OBTAINED

Extrameningeal lesions	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Lymph glands ...	—	—	—	—	1	—	1	—	—
					(100)		(100)		
Lung	2	—	—	2	—	—	2	2	1
	(100)			(100)			(40)	(40)	(20)
Miliary	—	—	—	1	2	3	3	—	1
				(16·6)	(33·4)	(50)	(75)		(25)
Bone and joint ...	—	—	—	—	—	—	1	—	—
							(100)		

TABLE 6—SHOWING THE INFLUENCE OF THE CONDITION OF THE FUNDUS ON THE RESULTS OBTAINED

Condition of fundus	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Abnormal	1	2	4	3	2	10	18	2	3
	(14)	(29)	(57)	(20)	(13)	(67)	(78)	(9)	(13)
Normal	6	—	2	7	2	1	10	4	3
	(75)		(25)	(70)	(20)	(10)	(59)	(23)	(18)

TABLE 7—SHOWING THE INFLUENCE OF C.S.F. SUGAR ON THE RESULTS OBTAINED

Sugar levels (mg.%) in c.s.f.	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
15-25	4	—	2	6	1	3	16	1	2
	(67)		(33)	(60)	(10)	(30)	(84)	(5)	(11)
25-30	1	1	1	2	1	2	4	1	1
	(33)	(33)	(34)	(40)	(20)	(40)	(67)	(17)	(16)
35 and over ...	2	1	3	2	2	6	7	4	3
	(33)	(17)	(50)	(20)	(20)	(60)	(50)	(29)	(21)

TABLE 8—SHOWING THE INFLUENCE OF C.S.F. PROTEINS ON THE RESULTS OBTAINED

Protein level (mg.%) in c.s.f.	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Above 200	4	—	2	6	1	2	9	1	3
	(67)		(33)	(66)	(11)	(23)	(69)	(8)	(23)
100-200	1	1	1	2	1	4	6	2	1
	(34)	(34)	(32)	(28)	(14)	(58)	(67)	(22)	(11)
Below 100	2	1	3	2	2	5	12	3	2
	(34)	(16)	(50)	(22)	(22)	(56)	(71)	(18)	(11)

TABLE 9—SHOWING THE INFLUENCE OF C.S.F. CHLORIDE LEVEL ON THE RESULTS OBTAINED

Chloride level (mg.%) in c.s.f.	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Less than 500 ...	1	—	5	2	2	6	3	2	2
	(16)		(84)	(20)	(20)	(60)	(42)	(28)	(50)
500-650	2	1	1	3	1	3	5	1	2
	(50)	(25)	(25)	(43)	(14)	(43)	(62)	(13)	(25)
Above 650	4	1	—	5	1	2	19	3	2
	(80)	(20)		(62)	(13)	(25)	(78)	(13)	(9)

TABLE 10—SHOWING THE INFLUENCE OF C.S.F. CELL COUNT ON THE RESULTS OBTAINED

No. of cells per c.mm. in c.s.f.	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
More than 100 ...	4	—	1	6	1	4	11	1	2
	(80)		(20)	(55)	(9)	(36)	(79)	(7)	(14)
50-100	2	1	3	3	2	5	6	1	1
	(33)	(17)	(50)	(30)	(20)	(50)	(75)	(12·5)	(12·5)
Less than 50 ...	1	1	2	1	1	2	10	4	3
	(25)	(25)	(50)	(25)	(25)	(50)	(59)	(23)	(18)

TABLE 11—SHOWING THE INFLUENCE OF DIFFERENTIAL CELL COUNT ON THE RESULTS OBTAINED

Type of cells in c.s.f.	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Mainly lymphocytes ...	6	1	4	9	2	8	26	6	4
	(55)	(9)	(36)	(47)	(10)	(43)	(72)	(16)	(12)
Mainly polymorphs ...	(1)	1	2	1	2	3	1	—	2
	(25)	(25)	(50)	(17)	(33)	(33)	(50)	—	(67)

TABLE 12—SHOWING THE INFLUENCE OF INITIAL TUBERCULIN TEST ON THE RESULTS OBTAINED

Tuberculin reaction	Chemotherapy (No./%)			I/T Streptomycin (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Negative	2 (22)	1 (11)	6 (25)	3 (25)	3 (25)	6 (50)	12 (55)	4 (18)	6 (27)
Positive	5 (83)	1 (17)	—	7 (54)	1 (8)	5 (38)	16 (89)	2 (11)	—

TABLE 13—SHOWING THE INFLUENCE OF SERIAL TUBERCULIN TESTING ON THE RESULTS OBTAINED

Tuberculin reaction	Chemotherapy (No./%)			Cortisone (No./%)		
	Recovered	Partly recovered	Expired	Recovered	Partly recovered	Expired
Negative to positive ...	2 (100)	—	—	12 (93)	1 (7)	—
Negative (same reaction) ...	—	1 (25)	3 (75)	—	3 (34)	6 (66)
Negative (downward reaction)	—	—	3 (100)	—	—	—

N.B.—The figures for I/T Streptomycin group were not available.

DISCUSSION

From the results presented in Tables 1-13, it can be observed that certain factors have a profound effect on the ultimate outcome of tuberculous meningitis. It would not be out of place to discuss the relative value of these factors:

1. Stage of the disease on admission (Table 3).
2. Stage of the mental condition of the patient on admission (Table 4).
3. Fundus changes on admission (Table 6).
4. C. S. F. chloride level on admission (Table 9).
5. Initial tuberculin reaction (Table 12).
6. Serial tuberculin reaction (Table 13).

On the other hand, our study did not reveal any significant effect of the following factors on the ultimate outcome of the disease:

1. The age of the patient (Table 1).
2. The sex of the patient (Table 2).
3. Coincident extrameningeal tuberculosis (Table 5).
4. C.S.F. sugar level (Table 7).
5. C.S.F. proteins level (Table 8).
6. C.S.F. total cell count (Table 10).
7. C.S.F. differential cell count (Table 11).

When these factors are analysed groupwise, certain important conclusions could be drawn in relation to the corticosteriod therapy.

It can be easily seen that whereas these factors influence the prognosis uniformly in all the groups, the extent of their influence varies in different groups. This is particularly true with the group of cases receiving prednisolone therapy. It was found that in spite of the advanced stage of the disease, fundus abnormalities, low C.S.F. chloride level and negative tuberculin reaction on admission, the prognosis in the steroid-treated cases, was not as bad as that of the other two groups.

On the other hand one finds that the magnitude of the effects of factors such as mental condition on admission and serial tuberculin reactions are almost the same in both groups of cases, e.g., those who had received chemotherapy, with or without intrathecal streptomycin and those who had received chemotherapy with corticosteroids.

The implications drawn from our study are simple. By far the best clinical guide to the

prognosis is the mental condition on admission. Serial Mantoux study is equally important. These factors are so important that, even in the face of corticosteriod therapy, the influence of these factors became evident. In addition, factors such as the stage of the disease on admission, fundus abnormalities, C.S.F. chloride level and the results of initial Mantoux reaction also exerted important influences. The effects of these factors can be overcome to a large extent with the aid of corticosteroids. Hence, corticosteroids may be indicated in the presence of these changes. On the other hand, factors such as age and sex of the patient, extrameningeal tuberculosis and sugar, protein and cell content of C.S.F. have no or minor influence on the prognosis.

Summary

The results obtained in 80 cases of tuberculous meningitis treated entirely with chemotherapy (15 cases), chemotherapy with intrathecal streptomycin (25 cases) and corticosteroids orally in addition to usual chemotherapy (40 cases) have been screened against a number of variable factors. The most important prognostic factors obtained were the stage of the disease and the mental condition of the patient on admission, abnormal findings in the fundus, the level of C.S.F. chlorides, and initial and serial tuberculin reactions. It was further found that tuberculous meningitis could be predicted to have entered an irreversible phase if the mental condition on admission could be staged as IIIb or IV, and the results of serial Mantoux testing were persistently negative. In such cases, the recovery rate even in corticosteroid treated group was not much different from that of other groups. On the other hand, while the prognosis was poorer in non-steroid treated cases in presence of fundus abnormalities, chloride level in C.S.F. below 500 mg. per cent and initial negative tuberculin reaction, under similar condition, it was better in the steroid treated cases. Thus these changes could well be said to indicate a reversible stage of the disease where corticosteroids could be used with advantage.

Acknowledgments

We wish to express our thanks to Prof. B. B. Bhatia and other members of the staff of the department of medicine for permitting us to study their cases. Our grateful thanks are also due to the members of the house staff for their cooperation during the study.

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PRACTITIONERS' SERIES

CHRONIC PULMONARY EMPHYSEMA

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Calcutta

Chronic pulmonary emphysema is an insidiously developing, progressively increasing, chronic, disabling disease of middle life, with distressing symptoms, which often taxes the therapeutic resources of the physician.

The major defects in the pathological physiology of this disease are in most instances a combination of bronchiolar obstruction due to bronchospasm, accumulated bronchial secretions, mucosal oedema, allergic or inflammatory, and overdistension, with loss of pulmonary elasticity. The result is increase in the work of breathing with reduction in pulmonary ventilation.

The aetiology of pulmonary emphysema is obscure. Chronic bronchitis is the most frequently associated condition. Bronchial asthma sometimes accompanies it. Recently it has been suggested that tobacco smoking may be an important predisposing cause of pulmonary emphysema.

Dyspnoea is the earliest, most characteristic and often the most distressing symptom. Cough is not only troublesome at times but damaging as well. It tends to increase the rate of development of pulmonary emphysema. Expectoration is variable. In most cases the sputum is mucoid and scanty. In cases with bronchial asthma it is thick, viscid and more difficult to expectorate than the sputum which is yellow or greenish purulent-looking in patients who have bronchial infection.

The hyperresonant percussion note, encroaching on the cardiac and hepatic dulness, the diminished breath sounds and the prolonged expiration are the characteristic signs. The rales and rhonchi, when present, indicate bronchitis and wheezing sounds bronchial asthma.

There are no definite radiological signs of chronic pulmonary emphysema but the following findings are suggestive, viz., widening of the rib spaces, elevation of the ribs and clavicles, depression of the diaphragm and increased translucency of the lung parenchyma.

A serious complication often encountered in patients with severe emphysema is respiratory acidosis or an increase in the carbon dioxide content of arterial (not venous) blood, being precipitated by infection, increased work of breathing or the administration of oxygen. The clinical manifestations are chiefly neurologic. Disturbances of consciousness such as drowsiness and coma are common. Muscular movements such as fine tremor

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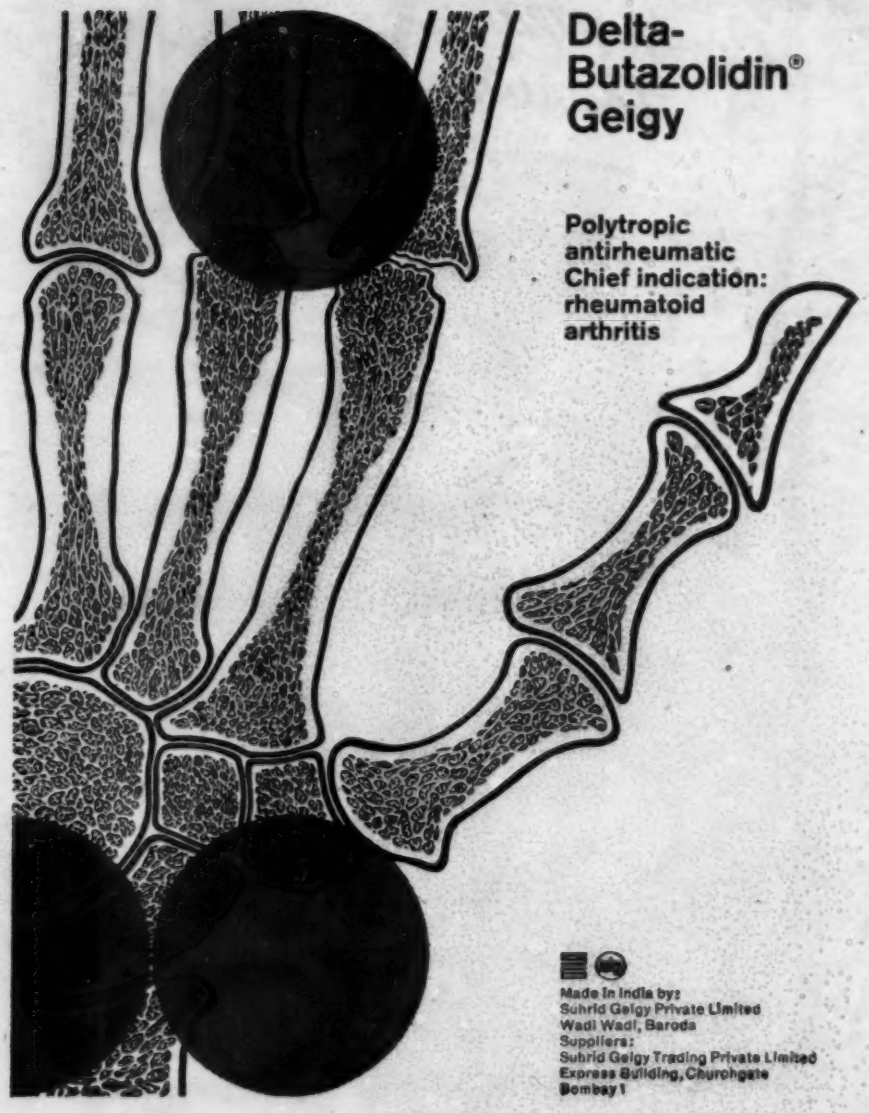

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Clinical Abstracts:

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Rustom Jal Vakil, M.D. (Lond.), F.R.C.P. (Lond.) D.T.M. & H. (Lond.), F.R.F.P.S. (G)

British Heart Journal 1949, 11, 350.

The most effective agent influencing the autonomous nervous system is "SERPINA", a combination of the alkaloids from the Indian plant Rauvolfia serpentina.

Prof. O. H. Arnold, M.D.

Ther. Gegenw., Germany 1952, 5, 167

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of the facial muscles and intermittent jerking of the fingers and arms are a characteristic feature of carbon dioxide narcosis. Cor pulmonale is a late complication of severe forms of chronic pulmonary emphysema. Clinically, the diagnosis is established by the demonstration of right heart enlargement with or without failure.

The diagnosis of advanced emphysema is evident from the physical examination of the patient but its detection in less advanced instances is often overlooked.

When the disease is well developed the pulmonary changes become irreversible. If bronchospasm is recognised before excessive hyperinflation is evident and treatment is undertaken before pulmonary respiratory decompensation has developed further progression of the disease may be arrested.

Prevention must start some years before the condition becomes disabling. Patients with bronchospasm of whatever cause should be diligently managed. Avoidance of respiratory irritants especially tobacco smoke and allergens must be enforced rigidly. In younger patients a careful search for extrinsic allergic factor to which the patient may be hypersensitive is to be made and if found he should be desensitised against it. In older people in the absence of extrinsic factor, treatment is to be made on the basis of bacterial allergy, with increasing doses of vaccine made from pyogenic micro-organisms obtained by sputum culture. If benefit is pronounced subsequent courses of treatment judiciously planned may avoid recurrence of symptoms. Chronic cough whatever the cause must be controlled. Every case of bronchitis must be treated properly and should not be allowed to persist long. The persistence of bronchial irritation is damaging in the long run ; the most likely damage being emphysema late in life. The patient with beginning emphysema should never be encouraged to lose weight even if he is obese. Fat within the abdomen assists in maintaining pressure on the diaphragm and hence facilitates expiration. Change of climate at a very early stage and change of occupation early in life should be considered.

In the absence of specific therapy, the treatment is partly supportive but mainly symptomatic.

Diet should be properly adjusted. Fats are poorly tolerated by patients with emphysema. Increased dyspnoea follows larger meals. The patient should take smaller meals at frequent intervals, five or six times a day.

Dusty occupations and industries producing irritant fumes are better avoided.

An equable climate, less than 2,000 feet above sea level, is recommended.

Bronchodilator therapy—Bronchospasm is more or less constantly present and needs relief. This is achieved in most patients by the use of bronchodilator drugs such as ephedrine, aminophylline and adrenaline. Ephedrine hydrochloride or sulphate in the usual dose of 16 to 60 mg. (¼ to 1 gr.), 3 times daily is often effective in mild cases of bronchospasm. For its prophylactic effect it may be continued for a long period. The drug may give rise to such side-effects as nervousness and sleeplessness and in older patients with prostatism urinary retention. It is contra-indicated in coronary artery disease. Tolerance to ephedrine is generally acquired fairly rapidly, its effectiveness often begins to decline by the end of the second week, interruption of treatment for 3 or 4 days restores its previous responsiveness. Aminophylline (theophylline ethylenediamine) affords prolonged relief in mild and moderate cases of bronchospasm in doses of 200 to 300 mg. (3 to 5 gr.), 3 times a day. It should be given after meals to avoid local side-action of gastric discomfort, nausea and vomiting. Tolerance to this drug develops more slowly than ephedrine. When the drug is long continued for its prophylactic effect a cumulative action may take place with an increase in adverse side-reactions. These can be prevented by temporarily withdrawing the drug for a few days. In severe cases of bronchospasm intravenous injection of 250 mg. (4 gr.) aminophylline in 10 ml. (150 minims) of water, repeated in 15 to 30 minutes if necessary, produces marked relief. This injection should be made very slowly over a period of 8 to 10 minutes to avoid serious cardiovascular reactions. Similar effect may be obtained by giving 600 mg. (10 gr.) of aminophylline dissolved in 30 ml. (1 oz.) of water as retention enema. Combinations of ephedrine with aminophylline may be used to increase their therapeutic effectiveness. Adrenaline may be injected subcutaneously in doses of 0·3 to 0·5 ml. (5 to 8 minims) of a 1 in 1000 solution, and repeated at intervals of 15 to 30 minutes, for the relief of acute paroxysmal attack of bronchospasm. Side-effects such as tremor, palpitation and excitability may occur. Tolerance may develop. This hypodermic medication has largely been supplanted by the oral inhalation of the drug. 0·1 ml. (2 minims) of a 1 in 100 solution is nebulised, by compressing the hand bulb of a De Vilbis no. 40 model nebuliser 4 to 6 times. The treatment may be repeated after a few minutes if required. Isoproterenol hydrochloride (isupren) is a more powerful bronchodilator than adrenaline. For the relief of bronchospasm it may be used as a sublingual tablet in 10 mg. (1/6 gr.) dose, not more than 3 times a day and at an interval of not

less than 3 hours. A common side-effect is tachycardia.

Expectorant therapy—Accumulation of bronchial secretions is common. Their presence provokes paroxysms of coughing which serve to accentuate the spasm of the tracheobronchial passages. Adequate elimination of the retained secretions has to be made by drugs as well as by drainage. Potassium iodide is one of the most valuable drugs employed in the treatment of pulmonary emphysema for relieving bronchial spasm and altering the thick tenacious sputum to a thinner secretion which is more easily expectorated. The dose recommended is 0·3 g. (5 gr.) 4 times daily after meals. Tolerance seldom develops. Side-effects include painful swelling of the salivary glands and skin eruptions requiring its withdrawal. It should not be given in pulmonary tuberculosis. In conjunction with potassium iodide, potassium arsenite solution (Fowler's solution) in doses of 0·2 ml. (3 minims) 3 times a day after meals has the reputation of relieving bronchospasm and is still employed with benefit in the treatment of chronic emphysema.

Bronchial drainage—This is facilitated by putting the patient to bed on an inclined plane with foot end elevated. It is carried out for one-half to one hour, 3 times daily. Postural drainage in order to be fully effective should be used in combination with bronchodilator and expectorant therapy.

Antibiotic therapy—Pulmonary emphysema is most frequently associated with chronic bronchitis which is predominantly of infectious origin. The infection, usually of the bronchioles, produces obstruction of their lumen by chronic inflammatory changes. An attempt should be made to control the infection which is possible in many cases by the administration of suitable antibiotics. The broad-spectrum antibiotics are the drugs of choice. Tetracycline (achromycin), chlortetracycline (aureomycin) and oxytetracycline (terramycin) are equally effective. Side-effects are diarrhoea, nausea and vomiting. Diarrhoea is most frequent with oxy- and least frequent with tetracycline itself. Nausea and vomiting can be avoided by giving the drug in small doses at frequent intervals. The acute exacerbations of the chronic infection are treated with tetracycline in doses of 250 mg., every 4 hours, by mouth for 7 to 10 days. For the treatment of the chronic infection tetracycline in doses of 500 mg. daily by mouth should be continued for several months. For the long-term treatment of chronic infectious bronchitis penicillin by mouth in a dosage of 1 million units twice daily may be given a trial, provided no allergic reaction to the drug has been previously found.

Hormonal therapy—ACTH and adrenal corticosteroids afford a more complete and continuous relief of bronchospasm than adrenaline and aminophylline. In addition, they exert anti-inflammatory as well as anti-allergic effects and reduce the mucosal oedema of the tracheobronchial air passages. The drug of choice is prednisone or prednisolone, both of which are equally effective and have the advantage over other members of this group in not causing salt and water retention. In acute exacerbations of bronchospasm in the course of chronic emphysema, 40 to 80 mg. prednisone a day, in 4 divided doses by mouth, gradually reduced to a daily maintenance dose of 10 to 20 mg. by the 14th day is recommended. Patients on maintenance therapy should be given intramuscular injection of corticotrophin gel 60 mg. every fortnight. Long continued medication may give rise to adverse reactions such as features resembling Cushing's syndrome. The drug is contra-indicated in peptic ulcer, diabetes mellitus and active pulmonary tuberculosis. Prednisone therapy should be reserved for those cases in which all previous therapeutic measures have been found to be inadequate to cope with the patient's dyspnoea. Even in long-term prednisone therapy the conventional bronchodilator and allied drugs should be continued in order to reduce the dosage of the steroid hormone to a minimum.

Oxygen therapy—Inhalation of oxygen is incated when severe dyspnoea, cyanosis and tachycardia occur during the course of the disease. It should be given continuously by nasal catheter and the flow rate gradually increased to avoid coma. The initial rate of administration is 1 litre per minute for the first 24 hours, with an increase of 1 litre per minute every other day until a flow rate of 7 litres per minute is attained. After a week or two with satisfactory restoration of cardio-respiratory compensation a gradual decrease in the administration of oxygen is made by lowering the flow, 1 litre per minute every other day, until the patient is returned to an air atmosphere. The rubber catheter should be greased every six hours. During oxygen therapy the administration of sedatives is withheld to avoid respiratory acidosis.

Sedation and relaxation—Mild sedation is desirable for inducing adequate rest. Chloral hydrate is the drug of choice. It may be given in doses of 0·5 to 1·0 g. (8 to 15 gr.) in 30 ml. (1 oz.) of water orally or by rectum every 8 to 12 hours. When heavy sedation becomes necessary to terminate intractable bronchospasm, 60 to 90 ml. (2 to 3 oz.) of ether dissolved in an equal amount of olive oil may be given rectally as a retention enema. This usually produces restful sleep with good relaxation for 2 to 4 hours. The patient

must be watched during sleep and his airway properly maintained. Morphine is dangerous, and barbiturates and paraldehyde are undesirable. Meperidine hydrochloride (pethidine) should be used when patients become refractory to other forms of bronchodilator therapy. Its use in proper dosage under close supervision is recommended as an excellent method of obtaining bronchial relaxation in special instances. The dose is 50 mg. intramuscularly, or 100 mg. orally. The drug may be repeated after 6 to 8 hours. The risk of causing addiction to the drug is a serious disadvantage.

Breathing exercises—As a result of impairment of elastic recoil of the lungs, serious impairment of diaphragmatic function takes place. Attempts should be made to enhance diaphragmatic breathing and restore the recoiling activity of the lung. Elevation of foot of the bed serves to shift the weight of the abdominal viscera against the diaphragm and thus to assist expiration. This may be employed during rest periods and at night. Lying supine, placing the hands on the abdomen and directing all attention to firm contraction of the rectus muscles during exhalation and trying to empty the lungs as completely as possible may help in the restoration of diaphragmatic function. This may be practised repeatedly to the point of mild fatigue followed by periods of rest, continuing for 15 to 30 minutes. A full inspiration is to be taken followed by a slow expiration against the pressure induced by constricting the lips (as in whistling). This is a convenient method of breathing exercise and may be repeated many times.

Other measures—The use of respirators, chiefly those providing intermittent positive pressure for inspiration, has been found to be effective in many instances.

CASE NOTES

LYMPHADENOPATHY IN A CASE OF INDIAN KALA-AZAR

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AND

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It is well recognised that variations occur in the clinical features of visceral leishmaniasis in different endemic areas of the world (Editorial, 1960). *Leishmania donovani* is demonstrable in the lymph glands by puncture in some cases of Chinese kala-azar and more frequently in African and Mediterranean forms of the disease but not in Indian cases. While lymphadenopathy may not be unusual or rare in African and Mediterranean forms of the disease, Indian kala-azar is usually not accompanied by enlargement of lymph glands (Napier, 1946). Christophers (1904) noted that lymph glands draining the normal skin did not show L.D. bodies but those draining a skin lesion showed scanty parasites. De (1934) found a small number of leishmania-containing reticulo-endothelial cells in a lymph gland draining an inflamed area in 1 out of 26 cases of kala-azar examined post mortem. He was of the opinion that parasitised histiocytes were usuallly not found in the lymph glands, submucous coat of the intestinal tract and organs and tissues other than the liver, spleen and the bone marrow unless there was a local inflammatory reaction. Recently an Indian child showing lymphadenopathy was admitted into the Hospital for Tropical Diseases, Calcutta, under the care of one of us (P.C.S.G.). The notes of this case are presented below :

Case Report

S., Indian female aged 10 years, a resident of Calcutta, was admitted into the Hospital for Tropical Diseases, Calcutta on 17-8-60 for irregular fever associated with weakness and emaciation, duration 6 months. She also complained of inability to see properly at night. About one year previous to the present attack of fever, the patient had suffered from fever that lasted for about 3 weeks. She was in fair health till the onset of the present attack of illness six months ago. The patient came from a family belonging to the low-income group and could afford only a little fish or milk once or twice a month at the most. Her food usually consisted of rice, *dal* soup, and a little vegetables.

There was no history of any other illness in the past and the family history was non-contributory.

On examination the patient was found emaciated (weight 37½ lb.), anaemic and slightly icteric. The conjunctiva was muddy in colour. She had a temperature of 100°F ; P/R was 120/26. The spleen was enlarged to 2″ and the liver to 3½″ below the costal margin. There were signs of bronchitis on the right side. No other abnormality was detected.

Blood : Haemoglobin 6·8 g./100 ml. ; RBC 2·3 million/c.mm. ; PCV 20 per cent ; WBC 4,250/c.mm. ; neutro. 56 per cent, lympho. 30 per cent, monocyte 14 per cent, eosinophil nil. *Aldehyde test* : + + + ; *Antimony test* : + ; *Complement fixation test for kala-azar :* serum anticomplementary ; *van den Bergh test :* direct delayed positive ; bilirubin content 1·5 mg. per cent. *Urine* : no abnormality. *Faeces* : giardia cysts and trichuris ova present. *Ilium puncture smear* : Leishman-Donovan bodies + + +.

Skiagram of the chest : no abnormality detected.

Course—The patient was put on daily injections of sodium stibogluconate on 23-8-60 and the fever came down to normal on 27-8-60. The patient appeared brighter but on close inspection it became apparent that she had rather prominent glands in the submaxillary region. On palpation it was found that the lymph glands in this region were enlarged. But there was no gross sepsis in the mouth. It was then noted that the posterior occipital gland on the left side and the axillary glands and the inguinal lymph glands on both sides were enlarged, as also the epitrochlear gland on the left side. This gland felt about ½" in diameter along the long axis. The other glands were about this size or smaller. The epitrochlear gland was punctured on 30-8-60 and the next day it was removed surgically for histological examination. The patient had 8 and 9 injections before the puncture and biopsy respectively were done.

The smear showed the presence of a fair number of leishmania form of *L. donovani*. The lymph gland was about 1 cm. in length and showed a haemorrhagic area in the capsule. On histological examination, there were found many leishmania-containing reticulo-endothelial cells and plasma cells besides lymphocytes and occasional eosinophils in the areas containing the parasitised histiocytes (Figs. 1 and 2, *vide* Plate). The gland showed mild degree of sinus catarrh and reticular cells containing pigment. Two small haemorrhagic areas caused by the puncture on the previous day were readily seen.

Culture of material aspirated from the lymph gland during puncture in N.N.N. medium showed heavy growth of leptomonads of *L.donovani*.

The patient was treated with two courses of injections of sodium antimony gluconate, the total dose being 66 ml. of solution containing 100 mg. of Sb per ml. She was discharged as cured on 23-10-60. She had gained almost 10 lb. in weight ; the spleen was not palpable and the liver was enlarged to ½" below the costal margin. The haemoglobin was 11·6 g. per 100 ml. of blood and WBC 9,700/c.mm. The lymph glands were no longer enlarged.

Comments

The present case showed enlargement of lymph glands in different parts of the body and it was possible to demonstrate L.D. bodies in lymph gland puncture smear and in histological sections. According to Napier (*loc. cit.*) the lymphatic glands are not sufficiently enlarged for puncture and when in an emaciated patient it is possible to grip the glands, the findings of lymph gland puncture are usually negative. It thus appears probable this is the first time that the parasite *Leishmania donovani* has been found in the lymph gland puncture in a case of Indian kala-azar. The histological changes in the lymph gland indicate that parasitised histiocytes were not accidentally caught up in a lymph sinus from an infected skin area because there were no skin lesions in the area drained by the gland. There was considerable local reaction of the type noted in leishmanial infection in other tissues and in the lymph glands, viz., presence of parasitised and non-parasitised histiocytes, lymphocytes and numerous plasma cells. The lymphadenopathy was thus essentially due to leishmanial infection.

The other features of interest in this case were marked hepatic enlargement associated with relatively slight enlargement of the spleen—an uncommon but not unknown finding and the presence of nightblindness which was apparently related to nutritional inadequacy in this case and possibly to hepatic disorder associated with kala-azar (Sen Gupta *et al*, 1952).

Summary

Mild but widespread lymphadenopathy was found in an Indian child, a permanent resident of Calcutta, suffering from kala-azar. *Leishmania donovani* could be demonstrated in the lymph gland puncture smear and there was evidence of leishmanial infection of the gland on histological examination. Though the parasite has been demonstrated in the lymph glands in African, Mediterranean and Chinese forms of the disease, it is probably the first time that it has been found in lymph gland puncture smear in Indian kala-azar.

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MULTIPLE PSEUDOCYSTIC TUBERCULOSIS OF BONE IN AN ADULT

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Multiple pseudocystic tuberculosis is an uncommon manifestation of tuberculosis of the bone. It is a distinct entity (Kominis, 1952) and should be differentiated from other tubercular involvements of the bone, e.g., solitary foci of diaphyseal tubercular osteitis, tuberculous dactylitis, Jungling's disease or osteitis tuberculosa multiplex cystoides. A confusion exists about the exact aetiology of Jungling's disease, for many workers consider it as a sarcoid manifestation (Ellis, 1940; Sweet and Abramson, 1941; Golding, 1950; Alexander and

Mansuy, 1950; Komnis, 1952; and Girdwood, 1953). Clinton Thomas and Young (1955) from Malaya listed 34 recorded cases from the literature and added one of theirs. It should be noted that all the above mentioned cases occurred in children and only one in an adolescent. Murray (1954) and De Pape (1954) reported additional cases. Karlen at Singapore (personal communication) reported about a few such cases in children.

The finding of such a rare condition in an adult has so far not been mentioned in the literature and hence such a case is reported here.

Case Report

P. S., male aged 30, working in a big industrial concern near Calcutta attended the orthopaedic department of the Medical College in April 1957 with complaints of pain, swelling and limitation of movement of the left wrist for one year. There was pain in the right shoulder for the last 6 months. He complained of weakness and deterioration of general health for the last one year. He was treated for rheumatism with salicylates, aspirin, vitamins and non-specific protein injections with no effect. There was no definite history of any contact with tuberculosis in the family.

On examination (24-5-57) the general build and nutrition of the patient was considered fair. The left wrist joint was swollen with tenderness and limitation of movement. Movement of the right shoulder was free but there was definite bony tenderness on the upper end of the right humerus. Skiagram of the left wrist showed localised cystic lesion in the carpus with soft tissue swelling (Fig. 1, *vide* Plate), while that of the right shoulder showed a few discrete small radiolucent cystic lesions in the upper end of the right humerus (Fig. 2, *vide* Plate), Skiagram of the lungs showed prominent bronchovascular markings and a definite localised lesion in the left upper lobe. The blood picture was normal and E.S.R. was 31/mm. 1st hour. The Mantoux test was positive in 1/10,000. Biopsy from carpus on 19-6-57 showed tuberculous granulation tissue with a few scattered giant cells.

During biopsy, curettage of the lesion was done properly and the wrist was fixed in a plaster of Paris casing in optimum position.

A full course of antituberculous treatment with streptomycin (90 g.), INH (500 × 100 mg.) and PAS was given for 9 months. A sling was given for the right shoulder.

The patient joined work with a painless and ankylosed wrist in optimum position and vastly improved general health after about a year of treatment. He came for regular check-up and is now quite fit.

Discussion

Multiple pseudocystic or cystic tuberculosis is a definite entity which is common in children, specially in this country. It is also common in other Afro-Asian countries where skeletal tuberculosis is rather common (Karlen, 1959).

Gross malnutrition, unhygienic living condition, poverty and associated infections may be the cause. The pathogenesis may be multifocal in origin. It may follow an initial bacteraemia from a primary infection, but it is difficult to prove it in man, though it is a constant finding in animal experiments (Wilson, 1933). Another explanation is that it may be due to an allergic state (Wallgren, 1948).

The arresting feature of this case, apart from its occurrence in an adult, was the small cystic involvement of the bones of two joints and the good response to treatment with antibiotics. Though *M. tuberculosis* was not isolated or cultured from the bone lesion or the sputum in this case, the lesion in the left lung, positive Mantoux test in high dilution, the histological findings of scanty multinucleated giant cells in necrotic granulation lesion of the bone and healing of bone and lung lesion with streptomycin, INH and PAS therapy are almost conclusive evidence of tuberculous aetiology in this case.

Acknowledgment

My thanks are due to Dr. S. C. Bose, Principal and Superintendent, Medical College Hospitals, Calcutta, for permission to use the hospital records of this case and to Professors K. Sarbadhikari, S. Mukherjee and S. De and members of the staff of the departments of orthopaedics, radiology and pathology for co-operation in the work.

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OSTEOGENESIS IMPERFECTA TARDA

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Osteogenesis imperfecta is a systemic disease of the mesenchymal tissues of the body. Some of the structures derived from the mesenchyna (sclerae, ligaments, bones, etc.) are developmentally inferior and exhibit the characteristic stigmata of the disease. The common stigmata are multiple fractures, blue sclerae and otosclerosis. Depending upon the age at which the first fracture occurs, there are three types described, viz., congenita, infanta and tarda. In tarda the first fracture is delayed till the age of 5 (Davel *et al*, 1954). Among other terms used for the tarda variety, osteopsathyrosis idiopathica is most common. There is usually a hereditary tendency and it is not unusual to find blue sclerae and otosclerosis in other family members, even in the absence of fractures.

Case Report

A female, aged 30, was admitted in M. Y. Hospital on 21-3-60 with complaints of pain in both lower limbs, curving of the legs and thighs, difficulty in walking and weakness—all of three years' duration. In the beginning she started having constant dull pain in both legs, more so on walking and in course of time she noticed bowing of legs which gradually increased to the present state. Initially, for about a year she could walk without a stick but later on only with its help. There was no history of trauma.

Past, personal and family histories—There was no history of significant illness in the past. Her periods were regular and she had 4 pregnancies. The first two children were stillborn at full term, while the third and fourth were premature deliveries at 5 months' gestation. There was no history of fracture or deformed bones in her family.

Examination—An average built female, weighing 80 lb., height 4 ft. 3½ inches, looked normal for her age. The skull and upper extremities were normal, but the lower extremities showed marked bowing of the thighs as well as of the legs, more marked in the left thigh and right leg. Sclerae of both eyes showed a definite bluish tinge. The pulse was 84 per minute, temperature 98°F, respiration 20 minute and blood pressure 120/80 mm. Hg. The spleen and the liver were not palpable and the respiratory and cardiovascular systems were normal. The ears were examined for evidence of otosclerosis, with negative result.

Investigations—Red blood cell count 4·2 million/c.mm., haemoglobin value 11·5 g. per cent; leucocyte count 6900/c.mm., with neutrophils 70 per cent and lymphocytes 30 per cent. Blood Kahn's test was negative. Urine showed faint trace of albumin but no abnormality microscopically. Stool examination showed no abnormality.

Blood urea was 20 mg. per cent; serum calcium 9 mg. per cent; inorganic phosphorus 2·3 mg. per cent; alkaline phosphatase 6·9 Bodansky units; plasma bicarbonates 27 mEq., total urinary calcium (on ordinary hospital diet) 160 mg. per cent.

The skiagrams of both femurs (Figs. 1 and 2, *vide* Plate), both tibiae and both fibulae (Fig. 3, *vide* Plate) showed fractures. The fracture of the right femur showed malunion. The pelvic cavity looked small and the ramus of the right pubic bone also showed a fracture. The skiagram of the skull showed prominent vascular markings; the skiagrams of chest, upper extremities, and the plain skiagram of the abdomen (for any evidence of renal calculi) did not show any abnormality.

Treatment—After the biochemical investigations the patient's diet was supplemented with two seers of milk daily. She was put on calcium lactate gr. 20, t.d.s., and massive injection of vitamin D_1 600,000 units, twice a week for about a month. There was no significant improvement in the clinical condition of the patient and the blood chemistry was repeated. This did not show any significant change.

Discussion

At the first glance the case appeared to be osteomalacia, because of the involvement of the lower extremities in a female of child-bearing age. But the clinical features, radiological characters, biochemistry and the follow-up of the case did not leave any doubt as to the diagnosis of osteogenesis imperfecta. The latter condition is not very uncommon and the tarda type is rather a rarity. Besides the rarity of the condition, certain features of this case are interesting. In most of the cases of osteogenesis imperfecta tarda described in the literature, the age of onset is rarely delayed beyond 16-17 (Brennemann, 1959; Scott and Stiris, 1953). In Bornebusch's (1940) review of 62 cases of osteogenesis imperfecta none of the cases showed the first fracture beyond the age of 16. The patient under review was healthy till 27 years of age, and then disability started gradually. The bad obstetrical history is possibly due to a small pelvis. Another interesting feature is the involvement of the bones of the lower extremities only. Bones of any part of the body may be affected, specially in congenita and infanta varieties, because all bones have similar chances of strain. However, in tarda type at this age it is very likely that only the bones which are weight-bearing and are put to maximum strain should be predominantly affected as observed in the present case. Jagermann (1931) described a similar case.

Summary

A case of osteogenesis imperfecta tarda is described. Symptoms started at an age as late as 27, and the bones of the lower extremities only showed fractures.

ACKNOWLEDGMENT

Thanks are due to Dr. G. C. Sepaha, Reader in Medicine, for guidance and to Dr. R. Bhattacharya, Superintendent, M. Y. Hospital, for permission to report the case.

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LATE ONSET OF FRIEDREICH'S ATAXIA

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Friedreich's ataxia is a heredofamilial, steadily progressive, degenerative disease affecting mainly the posterior columns, dorsal spinocerebellar tracts, pyramidal fibres and the posterior nerve roots of the spinal cord.

The full clinical syndrome is characterised by :

(a) An ataxia usually unaffected by closing the eyes, and associated with cerebellar signs such as nystagmus, scanning speech, dysdiadokokinesis and intention tremor. (b) Muscular weakness which may start as a fatigability and progress to paralysis. It is associated with loss of tone. (c) Impairment of deep sensibility with later loss of touch, pain and temperature. (d) Presence of congenital abnormalities such as pes cavus and kyphoscoliosis. Some have ascribed these to early pyramidal damage. (e) Less constant findings are optic atrophy, atrophy of hands and cardiac changes leading to congestive cardiac failure.

Incidence—The disease is a rarity with the incidence of 0·4 per cent in 15,000 cases seen at the National Neurological Hospital, London. It is hereditary and transmitted both by the male and the female, though the nature of transmission is much disputed, e.g., dominant (Dressel, 1917), recessive (Friedreich) and both (Brain, 1925). Sporadic appearance is also well known and was first shown by Blocg in 1887.

Males have a slight predominance over females, 94 out of 164 in Holmes series and 68 out of 115 in Socas series.

As a general rule, cases occur between the ages of 7 and 15 years. Younger cases have been described very often, even at birth (Biro, 1901; Wilson and Bruce, 1954) but cases of late onset are very occasional. Wilson and Bruce (1954) described one case at the age of 27 and Potts (1906) three cases at the ages of 28, 36 and 42.

CASE REPORT

Recently we had a case of late onset of symptoms. A male aged 30 was admitted for inability to walk and stand for the last two years. The complaints started with progressive weakness first noticed at the age of 26. For the last one year he was unable to sit up in bed. There was no family history of similar complaints on either side.

Clinical examination revealed a well-built male with normal intelligence. Pupils were normal, and fine horizontal nystagmus was present especially marked with upward fixation of the eyeball.

Tone and power were normal in both the upper limbs but the lower limbs showed marked loss of power especially in the adductors of thigh and the flexors of hip. Co-ordination was grossly impaired in all the four limbs and was not affected by closure of the eyes. Sensations were normal except for the slight loss of vibration and joint sensation in the lower limbs. Deep reflexes were absent and plantar reflex was extensor. Cerebellar signs like dysdiadokokinesis, past-pointing and rebound phenomenon were noticed. Spine showed scoliosis to the right. He had pes cavus.

The investigations showed normal haematological findings, normal C.S.F., negative V.D.R.L., normal x-ray of skull and normal x-ray of spine except for mild kyphoscoliosis. The E.C.G. showed left ventricular hypertrophy and depressed ST segment with inverted T wave.

COMMENT

As mentioned previously, late onset of Friedreich's ataxia is rare. It is known that an illness may cause the patient to postdate the onset of symptoms, e.g., one of Wilson's cases was unable to walk well in the dark since 'she could remember', yet attributed her symptoms to her menarche. The pathogenesis of the condition is believed to be an underdevelopment of the cord which allows decay of certain fibres at an earlier or later date. It is possible that a milder form of defect causes a later onset. The E.C.G. of the case described is typical of Friedreich's ataxia with no cardiac symptoms. The family members of the patient did not show any defect.

SUMMARY

A case of Friedreich's ataxia with late onset of symptoms and cardiac changes is reported.

ACKNOWLEDGMENT

The authors are deeply grateful to Dr. C. L. Modi, Dean, B. J. Medical College and Sassoon Hospitals, Poona for kindly allowing them to use the records of the case reported here.

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TUBERCULOUS MENINGITIS

Tuberculous meningitis has recorded a dramatic change in its outlook during recent years. It was considered to be uniformly fatal before 1948. A situation has now been reached when more than 90 per cent of the cases can be expected to survive[1].

The cardinal points in the pathology are: "a dense collar of tuberculous exudate surrounding the brain stem, spreading into the sylvian fissure, and filling the more anterior of basal cisterns"[2]. This exudate is responsible for the development of common cranial nerve palsies and communicating hydrocephalus. Obliterative endarteritis seen around the tuberculous foci can lead to infarction of the brain tissue.

Rich and McCordock[3] have focussed our attention on the role of focal caseous plaque in the meninges and tuberculoma in the substance of the brain, in the pathogenesis of tuberculous meningitis. By experimental, post-mortem, and pathological studies, they have proved that immediate miliary dissemination of the tubercle bacilli was not an essential feature in the causation of the disease. Other workers[4] have also confirmed their propositions.

Early diagnosis of tuberculous meningitis may be entirely clinical. The symptoms such as malaise, anorexia, fever, constipation, occasional headache and vomiting do not have anything specific about them except that they are unusually persistent[5]. Neck rigidity may be absent. However, obstinate constipation and some fullness of the fontanellae must be looked for in the early stages.

Cerebrospinal fluid changes include: a clear fluid which produces a cobweb coagulum, cell count 50-500/c.mm., and protein content between 80-900 mg./100 ml. The tendency to albuminocytological dissociation is more marked in this case than in other types of 'lymphocytic meningitis'. Differential cell count reveals monocytosis. As a rule, sugar content is reduced to below 40 mg. per cent. A fall in the level of fructose early in the disease has been claimed to be more diagnostic than that of total reducing substances[6]. The classical fall in the chloride level is a delayed phenomenon. A reassessment of Levinson's test[6] revealed it to be positive in over 80 per cent of the cases on admission. It could well be taken as a guide to the duration of treatment, because it would gradually become negative as the patients recovered. Study of partition of bromide between blood and C.S.F. has also been used as a diagnostic procedure. Increased penetrance of meninges to bromide ions has been demonstrated[7].

Isolation of tubercle bacilli from the C.S.F. has been highly unsatisfactory, especially in the hands of Indian workers[8]. Recently, application of electrophoresis of C.S.F. for the isolation of the bacilli has yielded hundred per cent results[9]. This method demands further study.

One of the investigations recently employed in tuberculous meningitis is air encephalography[10]. Sifontes *et al*[11] recommended it when the progress of the patient was unsatisfactory. A normal encephalogram usually indicated a better outlook with poor prognosis.

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Lorber[12] has recently drawn our attention to the role of intracranial calcification in the assessment of the prognosis of cases which have recovered from tuberculous meningitis. Calcification in the basal meninges was of the utmost importance as opposed to that of calcification in the substance of brain. Meningeal calcification was more frequently seen if the patient had entered the hospital in advanced stage, or if he was left with neurological sequelae. Six cases out of a total of 130, who relapsed later, had meningeal calcification, thereby giving indirect evidence of the survival of live tubercle bacilli in these areas. Besides these, other factors of prognostic significance are: extremes of age[13], stage of disease on admission, mental condition on admission[14], presence of neurological sequelae including optic atrophy, level of sugar and chloride[15] in the cerebrospinal fluid. A persistently negative tuberculin reaction was a bad omen[16]. Lymphocyte and monocyte ratio in the peripheral blood, if below 5, has been found to be associated unusually with bad prognosis[17].

Chemotherapy alone in mild cases can now effect a recovery in over 80 per cent cases. The place of intrathecal streptomycin, however, still remains to be decided. The Scottish Committee[18] has voted against such form of therapy but their conclusions are not beyond criticism. It has been rightly pointed out by Lorber[19] that no double blind trial has as yet been conducted on this aspect of the problem.

Intrathecal use of streptokinase and streptodornase to dissolve out thick exudates may do more harm than good[20]. Smith *et al*[21] have advocated the use of intrathecal P.P.D. to achieve the same objective. This agent could be used only if (a) diagnosis was confirmed by isolation of T.B. or a recent miliary spread, (b) the patient responded as a positive reactor to 100 T.U. of P.P.D. given intracutaneously, and (c) no signs of cord compression were present. The results achieved by these workers have been fairly good, but one has to remember that extensive hospital facilities for neurosurgery must be available before this form of therapy can be brought into practice.

On the other hand, corticosteroids have also been advocated in the treatment of this condition. The results obtained in corticosteroid-treated cases have been extremely encouraging, so much so that in one series[22] 97 per cent survival rate has been reported. Intrathecal use of hydrocortisone has also been advocated[23], but as yet no controlled trial to confirm or refute its utility has been conducted. It is important to remember that electrolyte disturbances similar to those met with in Addisonian crisis have been described in tuberculous meningitis[24]. A case with severe hyponatraemia and hypokalaemia has been described[25]. This case, responded satisfactorily only when corticosteroids were added to the therapeutic regimen. This might very well form another pharmacological basis, besides the anti-inflammatory, anti-allergic and replacement actions, for the use of corticosteroids. However, corticosteroid therapy is not without hazards[26].

Optic atrophy following tuberculous meningitis has been one of the dreaded complications. Nicotinic acid may effect a recovery in some of the cases[27, 28].

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PRESIDENT'S COLUMN

SHORTAGE OF DOCTORS

Dr. H. N. Shivapuri, President, Indian Medical Association, issued a letter to the Editor, The Times of India, *on 11-11-60 in reply to a leading article published in that paper a few days earlier. The following is from Dr. Shivapuri's letter published in* The Times of India *dated 18-11-60 under Readers' Views*—ED., *J. Indian M. A.*

There is said to be a shortage of doctors in our country and on that plea, propaganda has been launched for restarting the Licentiate Course. It is not clear how the restarting of this course will reduce the so-called shortage of doctors in our country. Even if the course is restarted, it will not produce any doctors before a minimum period of four years, possibly more, as before a Licentiate Course is started proper institutions will have to be built and necessary teaching staff and equipment provided. This means time and additional expense ; and by the time these institutions are ready to produce licentiates, the large number of medical colleges (about sixty) that have already been started will also be producing medical graduates in large numbers.

The propaganda for restarting the Licenciate Course is, therefore, no solution to the problem of vacant rural dispensaries. Where then lies the remedy? Let us for a moment consider why the dispensaries are lying vacant. There is no dearth of doctors even today. The main problem is the problem of wages. The various State Governments do not want to pay a living wage to the medical man. A medical course is long and very expensive. A medical graduate is usually about twenty-seven or twenty-eight years' old when he is ready to join service. In other services, the usual age is about twenty-three or twenty-four. Therefore the paying of a proper wage to a medical man is very important.

There is no guarantee that even a licentiate will go to a rural area under the present conditions of service. There are no living quarters and no educational facilities for his children. The real reason for rural dispensaries lying vacant is not a shortage of doctors but the absence of proper facilities and poor wages. If these can be adjusted there will be no shortage of doctors.

* * * *

Another important reason for the dispensaries remaining vacant is bad planning and poor co-ordination * * *. The Indian Medical Association, the All India Licentiates' Medical Association and the Medical Council of India are all unanimously opposed to the revival of the Licentiate Course and will oppose such a retrograde step tooth and nail. There is no such course anywhere else in the civilised world. A rural life is no less valuable than an urban one and there is no justification for sending a less qualified doctor to rural areas.

GENERAL PRACTICE DIGEST

TREATMENT OF POLIOMYELITIS

The medical treatment of poliomyelitis is best discussed under three headings, viz., (1) immunisation against poliomyelitis, (2) treatment of non-paralytic attack, and (3) treatment of paralytic poliomyelitis.

IMMUNISATION AGAINST POLIOMYELITIS

The Salk vaccine contains all three types of virus which have been grown on kidney tissues of the monkey and inactivated by formalin. It contains a mineral oil adjuvant. The vaccine is administered in three doses—the first and second doses are given at intervals of a month and a third booster dose six months later.

The Salk vaccine is expensive and needs three injections. It does not prevent alimentary infections (which may infect others) and is ineffective in halting an epidemic. In this respect Koprowski's avirulent type II (Lansing) vaccine, and Cox's avirulent trivalent vaccine offer improvements. Both of them can be given orally; they are inexpensive and can be given in one dose, even during an epidemic. They have been administered safely to about half a million children in Mexico, U.S.S.R. and Malay.

TREATMENT OF NON-PARALYTIC POLIOMYELITIS

During an epidemic of poliomyelitis, all children with trivial cold, fever and headache should be put to bed at once and this should be continued for one week after the temperature has come down to normal. The patients and their immediate contact should try to avoid physical exercise, operations and all sorts of infections for one month. These measures are intended to reduce the chance of non-paralytic cases developing paralysis.

TREATMENT OF PARALYTIC POLIOMYELITIS

I. TREATMENT OF THE UNCOMPLICATED CASE :

The patient should be confined to bed at the earliest evidence of paralysis, if this has not already been done. A case should not be transferred to a hospital unless there is difficulty in breathing or swallowing or involvement of the deltoid (which presages involvement of the diaphragm) or there are evidences of a severe attack, such as continuous high fever or toxaemia.

The limbs should be put in the neutral position by means of pillows and the feet should be dorsiflexed with the help of boots with a cross bar, or a board at the foot of the bed. Children require light splints, otherwise they may not rest. To avoid claw hand, the fingers should be folded over a ball in the palm of the hand, the the thumb being kept at right angles to the palm. If the abdominal and trunk muscles are paralysed, a bed with a hard board and good alignment of the trunk is essential.

Pain and spasm of muscles can be relieved by repeated small movements of the limbs, by supporting pillows and by hot wet packs applied four times a day. Aspirin, gr. 10, four times a day, is very useful in relieving the pain.

Adhesions are likely to form readily in immobilised joints. Therefore, after the first week, each of the joints should be moved through its full range of movement—at first once and later twice a day. The application of hot packs before performing the passive movements considerably relieves any pain and spasm which might restrict such movements.

II. MANAGEMENT OF COMPLICATIONS :

(a) *Diaphragmatic and intercostal paralysis*—If the patient becomes breathless on talking or he cannot count up to twenty in one breath, he needs a respirator. Other indications for a respirator are a rising blood pressure, a vital capacity below 50 per cent, a reduction of the tidal air to 250 ml. at each breath, and a value of alveolar CO_2 over 60 per cent.

The collar of the patient in a respirator should not be very tight, otherwise venous congestion will be produced. The rate should be adjusted at 18 per minute and the pressures at +10 and −10. The patient should be taken out of the respirator four times a day for attention to his back and toilet, the spine hyperextended and flexed and forcible deep breaths given to prevent fixation of the chest and back. Artificial respiration is necessary during the patient's stay outside the respirator.

The hazards of the respirator includes atelectasis which occurs in 25 per cent of cases, and is manifested by a shift of the apex beat. Atelectasis is treated by bronchoscopic aspirations. Other risk is an excessive water and electrolyte loss due to the heat inside the respirator.

(b) *Bulbar paralysis*—Adequate postural drainage, tube feeding, and maintenance of the respiration are important considerations in this condition. The patient should lie in the semi-prone position with the foot end of the bed raised by at least 18 inches. The mouth could be kept open, so that saliva can trickle out, and suction applied from time to time to clear the air passages.

During the first 24 hours or until the temperature becomes normal, intravenous fluids are necessary. For a child of 30 kg. an adequate intake will require 1,800 ml. of 10 per cent glucose in water, 600 ml. of 0·85 per cent sodium chloride in water or 1,200 ml. of 0·3 per cent potassium chloride in 10 per cent glucose. A total of 3,600 ml. of water, 300 g. of glucose, 48 mEq. of potassium and 96 mEq. of sodium should be given in 24 hours. The rate of administration of the fluid will depend on the specific gravity of urine. The bedside estimation of chlorides by Fante's test (on an optimum 5 to 7 g of chlorides per litre per day should be excreted) should be done. After 7 to 8 days, administration of intravenous fluids should be replaced by tube feeding.

Tracheotomy is indicated when cases with difficulty in swallowing develop difficulty in breathing. This is due to bilateral adductor paralysis or combined bulbar and diaphragmatic paralysis. The latter type also requires cuffed tracheotomy tube, with packing of the nasopharynx —respiration being maintained either by a tank respirator or by an intermittent positive pressure one. The cuff of the tracheotomy tube has to be relaxed every hour throughout the 24 hours, or a stricture of the trachea may occur.

The main disadvantage of prolonged intermittent positive pressure respiration is a fall in the venous return and circulatory embarrassment. This is avoided by using a respirator which introduces a negative phase.

The tracheotomy tube may give rise to a polyp. This should be looked for in every case.

(c) *Fluids and electrolyte disturbances*—In cases treated in a respirator, the insensible loss of water may increase to 3,000 ml. (normal 1,500 ml.) due to fever and hyper-ventilation. Polyuria of low specific gravity may double the normal basic output of 1,000 ml. in 24 hours. Excessive sweating may occur in a tank respirator, with fluid loss of 2,000 ml. a day. There may also be vomiting resulting in depletion of sodium, potassium and chloride. Fluids and electrolytes can be replaced by intravenous or oral route.

The basic requirement for a child weighing 30 kg. has been mentioned above, but extras should be added for vomiting. The specific gravity of urine should be 1010—1020, with a chloride content of 5—7 g. per litre.

Overhydration produces headache, vomiting, convulsions and polyuria, and underhydration produces pyrexia. Potassium deficit causes oligaemia, muscle weakness and electrocardiographic changes. Sodium and chloride retention produces oedema, and a drop in the intracellular sodium.

(d) *Central disturbances of respiration*—Irregular, rapid, shallow, incoordinated respiration or grouped breaths with an apnoeic panse indicate a central disturbance of respiration, and may result from anoxia or extension of bulbar poliomyelitis. Treatment includes tracheotomy, followed by artificial respiration with oxygen. The centre usually recovers and artificial respiration can be discontinued after a week.

(e) *Aspiration atelectasis and associated infection*—These may be prevented and treated by a semi-prone position, tapping the chest, frequent changes of posture, suction and bronchoscopic toilet. Oxygen and antibiotics should be added to the above treatment schedule.

(f) *Pulmonary oedema*—This results from cardiac damage from anoxia or a virus infection of the heart muscle. As blood transfusions and large amounts of intravenous fluid precipitate pulmonary oedema, these should be avoided, if possible. Pulmonary oedema is treated with adequate oxygenation, preferably with one of the anti-foaming agents and intravenous aminophylline, 0·1 to 0·5 g. in 50 per cent glucose.

(g) *Asphyxia, bradycardia, hypertension and hypotension*—These are due to involvement of the medullary centres by anoxia or by the poliomyelitis virus.

Hypoxia produces bronchial constriction. This can be relieved by ephedrine sulphate by mouth and aminophylline parenterally in 50 per cent glucose. A clear airway and arrangement for a respirator are essential.

Bradycardia is self-limited and requires no treatment. If it is associated with other evidences of excessive parasympathetic activity, a vagal blocking drug (banthine or antrenyl) may be given intravenously and repeated in 50

minutes. For associated gastro-intestinal bleeding a stomach tube has to be passed and necessary drug is given through the tube. Atropine is contra-indicated as it thickens the secretions.

Haematemesis requires blood transfusion.

Hypertension and hypotension may be produced by brain-stem poliomyelitis—the pressure rising and falling precipitously. Hypertension is treated by parenteral serpasil, 80 to 125 mg. in 24 hours—one quarter of the dose being administered immediately. Ganglion blocking drugs are not used because the hypertension is often followed by a drop of pressure. Hypotension and peripheral circulatory failure should be treated promptly by a noradrenaline drip, 4 mg. in 500 ml. of 10 per cent glucose. Such an infusion may be required for days or weeks. Blood transfusions should not be used for peripheral failure of central origin, because pulmonary oedema may ensue.

(h) *Myocarditis*—Impaired cardiac function may be produced by factors like anoxia, hypokalaemia, hyper calcaemia, viral invasion of the myocardium and hypotension. This is manifested by muffling of the heart sounds, triple rhythm and electrocardiographic changes.

The patient with poliomyelitis is very sensitive to digitalis. Hence digitalisation should be undertaken with caution—a small initial dose should be gradually increased.

—From an article by E. P. Bharucha, *Indian J. Pediat.* **27**: 331, 1960.

CURRENT MEDICAL LITERATURE

Response to Treatment of Chronic Cardiac Failure

Many studies have been made of the haemodynamic disturbances in congestive heart-failure. Selzer and McCaughey have now investigated cardiac function in patients in whom heart-failure had been brought under control by medical treatment. 50 patients with disease of the left ventricle were studied. In the majority ischaemic heart-disease and hypertension were the underlying disorders; but a minority had aortic-valve disease, and 3 had "idiopathic myocarditis". The patients were studied by cardiac catheterisation when symptoms and signs of cardiac failure had disappeared and a full response to diuretics had been obtained.

The circulatory abnormalities fell broadly into two categories. In one, the indirect left atrial pressure (pulmonary capillary venous "wedge" pressure) was normal at rest, indicating a normal left ventricular end-diastolic filling pressure; while in the other the indirect left atrial pressure at rest was persistently raised, suggesting persistence of left ventricular failure. Where the left atrial pressure was normal the cardiac output was either normal (judged by an arteriovenous difference of less than 5·5 vol. per cent) or reduced. With the output normal the pulmonary arterial and right atrial pressures were usually also normal or nearly so; but when the output was low the pulmonary vascular resistance was usually high. The response to exercise was almost always abnormal, the pulmonary arterial pressure rising without a commensurate rise in output—indicating an increase in pulmonary vascular resistance with effort.

The patients with a persistently raised left atrial pressure after treatment were regarded as having chronic left ventricular failure. In some of them the cardiac output was normal or close to normal, and mild passive pulmonary arterial hypertension was present, pulmonary vascular resistance being normal. But in others the pulmonary vascular resistance was raised, sometimes grossly, and in such cases increased right atrial pressure suggested right in addition to left ventricular failure. In all 50 cases the cardiac output was inversely related to pulmonary vascular resistance, and when the latter exceeded 300 dynes/sec./cm.$^{-5}$ (3·7 units), the cardiac index was less than 2·5 l./min./m.

Selzer and McCaughey conclude that failure of the left ventricle responds to therapy in three ways: first, the cardiac output, the ventricular filling pressure, and therefore the left atrial pressure, return to normal at rest, but the response to exercise remains abnormal indicating impaired functional reserve; secondly, the ventricular filling pressure returns to normal at rest but the cardiac output remains low; thirdly, the resting cardiac output returns to normal but the left ventricular filling pressure and left atrial pressure remain raised at rest. In this third group the pulmonary vascular resistance rises and may contribute to right ventricular failure and tricuspid insufficiency, which tend further to reduce the cardiac output. Probably the left atrial and pulmonary venous hypertension are followed by pulmonary arteriolar vasoconstriction, as has been postulated in mitral stenosis. In the cases studied the nature and severity of the haemodynamic disturbances were unrelated to the aetiology, duration of symptoms, and severity of cardiac failure; and, as might be expected, many patients with satisfactory clinical recovery from failure, and with little disability, had severe haemodynamic abnormalities.

These findings suggest that restriction of effort, cardiotonic drugs, and possibly diuretics should be continued after apparently full clinical recovery; for the myocardium requires additional support to cope with the extra work entailed by physical exertion. The importance of effectively reducing left ventricular end-diastolic pressure by adequate treatment is obvious for this will prevent an increase in pulmonary venous pressure, pulmonary oedema, and pulmonary arteriolar constriction, with reduced cardiac output and eventually right ventricular failure. Digitalis, by its stimulant action on the ventricular myocardium, has been shown to improve myocardial function and reduce left ventricular filling pressure. Consequently left atrial and pulmonary venous pressures fall, and the raised pulmonary vascular resistance is reduced, so that the cardiac output increases. It is clear from this work that digitalis should be continued after clinical recovery from left ventricular failure in sinus rhythm; and it is worth remembering that theophylline ethylene diamine (which can be given in suppository form, or by mouth as choline theophyllinate) may also be of value in improving myocardial function. (Annotation, *Lancet*, **2**: 357, 1960).

Flexor-Tendon Ruptures in the Forearm and Hand

BOYES, J. H., WILSON, J. N. AND SMITH, J. W. (*J. Bone & Jt. Surg.*, **42A** : 637, 1960) from New York give in the following lines the summary of their observations :

Eighty ruptures of flexor tendons, occurring over a thirteen-year period in the fingers in seventy-eight patients, were studied. The tendons involved were the flexor digitorum profundus, flexor digitorum sublimis, and flexor pollicis longus.

The majority of ruptures occurred in young patients, suggesting that degenerative changes accompanying ageing were not directly responsible for rupture.

The force producing the rupture was often slight, indicating that some underlying factors had already predisposed the tendon to rupture.

Flexor-tendon ruptures were found to occur most frequently after crushing injuries, lacerations, hyperextension injuries, and as the result of forced flexion against resistance. In 67·9 per cent of the tendons, these ruptures were found at the insertion or musculotendinous junction. In 31·9 per cent, they occurred within the tendon substance.

Hyperextension injuries most often produced ruptures at the insertion. Lacerations and crushing injuries were also frequently followed by rupture of a flexor tendon at its insertion or elsewhere within the finger. Ruptures resulting from forced flexion against resistance produced injuries at the tendon insertion, except in those instances where the tensile strength of the tendon had been decreased by pathological changes. In 20 per cent of all the ruptures, pathological changes in the tendon accounted for rupture and in nearly all of the tendons with these changes the ruptures occurred in the palm, carpal tunnel, or wrist.

It is emphasised that surgical treatment is often unnecessary. Surgery is essential only when flexor function has been severely compromised or lost.

Basic Surgical Principles of Ureteral Repair

WEAVER, R. G. (*Surg. Gynec. & Obst.*, **110** : 594, 1960) from the Departments of Anatomy and Surgery, College of Medicine, University of Utah, Salt Lake City, in presenting the ten surgical principles governing the diseased or injured ureter writes :

The laboratory background has been explained and the surgical application of these principles represent a composite of the work done by the investigators and urologists.

The principles involved include the following : (1) Immobilise the healing ureter. (2) Leave the splint *in situ* long enough for healing to take place . (3) Use the type of splint which is applicable to the case. (4) Do not substitute a splint for a carefully placed suture line. (5) Use different methods of treating the defects for different sites. (6) Use of fat cushion in the presence of periureteral inflammation. (7) Do not transect the ureter. (8) The complete defect heals poorly. (9) Suture the ureter loosely with fine sutures. (10) Drain the operative site.

Endocrine Ophthalmoplegia

SCHULTZ, R. O. VAN ALLEN, M. W. AND BLODI, F. C. (*A.M.A. Arch. Ophth.*, **63** : 217, 1960) from the Departments of Ophthalmology and Neurology, State University of Iowa and the Sections of Ophthalmology and Neurology, Veterans Administration Hospital, Iowa City, write that they have found ophthalmoplegia of varying degrees in 14 per cent of 165 patients with thyrotoxicosis.

The extraocular muscle weakness appears to be related to thyrotoxicosis rather than to exophthalmos, both in time of onset and in response to therapy. Approximately 50 per cent of the patients with ophthalmoplegia experienced return of normal eye movements following control of thyrotoxicosis, and this occurred in spite of the fact that proptosis increased following the use of radioactive iodine.

None of our observations support the contention that the ophthalmoplegia of thyrotoxicosis is secondary to exophthalmos *per se*.

The superior rectus and inferior oblique muscles are most prominently involved in thyrotoxic ophthalmoplegia.

Myasthenic symptoms in addition to the ocular manifestations occurred in 3 of 23 patients with ophthalmoplegia and thyrotoxicosis. The ocular muscle weakness, as well as the generalised signs of myasthenia disappeared with control of the thyrotoxic state.

Electromyographic studies in thyrotoxic ophthalmoplegia were consistent with true myopathy.

Electromyography done during the acute phase of nontoxic, congestive exophthalmos, in one case, showed a neuropathic pattern. A biopsy of the external rectus muscle revealed essentially normal muscle tissue.

Ocular Findings in Cystic Fibrosis of the Pancreas

BRUCE, G. M., DENNING, C. R. AND SPALTER, H. F. (*A.M.A. Arch. Ophthal.*, **63** : 391, 1960) from the Institute of Ophthalmology and the Babies Hospital of the Presbyterian Hospital and the Departments of Ophthalmology and Pediatrics, Columbia University, College of Physicians and Surgeons, in reporting on ophthalmologic abnormalities in 24 cases of cystic fibrosis of the pancreas with moderate to severe pulmonary involvement observe :

All patients included in this study revealed characteristic findings in the ocular fundi, which comprise varying degrees of venous engorgement and oedema of the disc, marked in some instances by haemorrhages and in others by cystic changes in the macula.

Factors common to all patients studied were severe pulmonary insufficiency and elevation of gamma globulin content of serum and cerebrospinal fluid. Six of fifteen patients examined had elevated venous pressure, three of ten patients examined had elevated cerebrospinal fluid pressure. Only 2 of 24 patients had a significant degree of carbon dioxide retention. Seven of eighteen patients had a prolonged prothrombin time. Normal amounts of macroglobulins were present in all sera.

The immediate aetiologic and prognostic significance of these findings is being investigated further.

CURRENT TOPIC

PUBLIC HEALTH EDUCATION AND RADIATION HAZARDS

M. G. CANDAU

*Director-General, World Health Organisation**

There exists today in many countries a remarkable interest in health questions related to ionising radiation. And in the face of modern developments this concern is easily understandable. Radiation, radio-activity and atomic energy present a very great and rapidly growing challenge in public health.

What is often not realised, however, is that more is known today about ionising radiation than about almost any other important environmental or industrial hazard. A great deal of experience and information on the effects of radiation on human beings has been accumulated. X-rays and radium have been used in medicine since the turn of the century; and a great deal has been learned from other sources, such as through the unfortunate early experiences with radium in the luminising industry.

Birth of Atomic Energy

Against this background of knowledge, deepened by research in radiobiology and the metabolism of natural and accelerator-produced radionuclides, atomic energy was born some 18 years ago. From the health point of view, the birth of the atomic energy industry was unique, because from the very beginning there existed a realistic appreciation of the very great potential health hazards which would accompany it. It was realised that large amounts of radiation would be involved, that new types of radioactive elements would be produced and, more important, that the creation of vast quantities of radioactivity would be associated with uranium fission on a large scale. There was an awareness that exceedingly great precautions would have to be taken for health protection.

The new atomic energy developments stimulated a marked intensification of studies on the biological effects of radiation and the behaviour of radioactive materials in the environment and in the body. And so over the years we have built up a relatively substantial understanding of radiation health problems.

Even in the realm of a very low level irradiation, where our knowledge is most meagre, we are not at a complete loss. We know that since the beginning of his life on this planet man has been bathed in cosmic radiation and exposed to the gamma rays from natural radioactivity in the earth's crust; he has been inhaling the natural radioactivity of the atmosphere, and been bombarded by the alpha and beta particles and gamma radiation from the radioactive elements of his own body tissues. We know something about the intensity and nature of this natural radiation and its fluctuations. And this provides us with a valuable yardstick with which to compare our newer man-made sources of radiation exposure.

We are keenly aware of our need for much more complete and fundamental knowledge in the field. That we can in fact evaluate and take into account radiation hazards more adequately than we can many others connected with modern life is an indication not of any truly deep understanding of radiation but of our ignorance in regard to other everyday industrial and environmental hazards.

Widespread Use of Radiation

If the radiation problem with which we are concerned stemmed simply from atomic energy activities, the matter would not be so difficult. Actually, the public health concern is very much wider. While it includes atomic energy, it is related more generally to the widespread, diffuse and rapidly expanding use of x-rays, radioactive material and other sources of ionising radiation in all sorts of activities—in agriculture, mining, atomic energy, industry, medicine, college and universities and various other enterprises both large and small. The use of radiation and radioactivity is part of everyday life in many countries and is becoming more so all the time.

Because the problems are varied, many different disciplines are involved. The disposal of radioactive waste from laboratories, hospitals, industries and atomic energy installations clearly involves sanitary engineering. Accidents in industrial and atomic energy installations or in the transport of radioactive materials may provide urgent and very complex public health problems, calling for assistance from many groups of workers.

Economically valuable uses of radioactivity cannot be safely introduced or exploited in any country unless there is sound preparation of health safeguards, and unless the public health authorities and personnel have the awareness, competence and preparation to fulfil their responsibilities in both routine and emergency situations. The same is true for atomic energy developments. It has become necessary today for physicians, public health officers, nurses, sanitary engineers, health educationists, industrial hygienists and other groups concerned with health to know much more about radioactivity, ionising radiation and atomic energy.

Public Health Education

How is public health education and training in this field best handled? Surely no single way is adequate. One of the most characteristic features of radiation health is the degree to which the physical and biological sciences must together be brought to bear on the questions involved. This interdisciplinary nature of the field presents special difficulties in connection with training.

The person broadly competent in radiation health must be soundly based in physics, biology, chemistry and medicine. It is unrealistic at the moment to ask

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* Text of an address at a meeting held by the Royal Society of Health, London, and reproduced from the *Journal of the Royal Society of Health* (**80**: 3, 1960).

for large numbers of such individuals in any country. Access to such competence, however, is necessary. The bulk of radiation health work in practice is carried out by persons from different training backgrounds. This is an area characterised by close co-operation and team work between physicians, engineers and physicists. It is a composite field, made up of many approaches and activities, dealing with a wide spectrum of problems. A very real structure and unity does exist, however, and nowhere is this more clear than in public health.

The educational roots lie far back in the undergraduate curriculum, where scientific foundations are laid and where any artificial division between the physical sciences and biology must be bridged. Radiation medicine and radiation pathology should be taken fully into account during the course of professional training in any modern medical school and the graduating doctor should have a basic understanding of these questions.

Special training is required at the post-graduate level. It is here that both the details and the broad public health view must be developed. Here radiation health takes real form as its various aspects fall into perspective.

One of the constantly recurring complaints about the medical curriculum is that it is overcrowded and lacking in coherence. The enormous increase during the past generation in the subject matter of scientific medicine has raised issues which are difficult to solve. In the undergraduate curriculum, there is a potential danger that the introduction of too many subjects will lead the student into acquiring a mere smattering of knowledge without any depth or real understanding.

The selection of subjects is increasingly difficult, because each expert teacher is naturally anxious to secure as much time as possible to present his own material, and is not inclined to show any great solicitude for his colleagues who are equally anxious to push their own subjects. Yet this knot must somehow be united and a compromise reached. What are the principles on which we should progress towards this aim?

The first point is that new knowledge, which is of importance to the health of people in the future and can be effectively applied to prevent risks, must somehow receive priority. In many academic institutions, the undergraduate curriculum is admittedly overcrowded with detail; but it is not enough simply to meet these difficulties by reducing the quantity of facts poured into the student from all sides.

The second point is that it would be no solution merely to withdraw the teaching of the basic sciences and introduce a whole series of disconnected bedtime stories about antibiotics, cardiac surgery or the hazards of radiation. One of the essential requirements is to link every subject of vital importance with one at least of the fundamental sciences. In other words, teaching must be structurally connected, so that each subject has a foundation on which it can stand.

Two Special Courses

The World Health Organisation has given specific attention to the question of training in radiation health. I would like to refer here especially to two studies of expert committees in the education field: one (*Wld. Hlth. Org. techn. Rep. Ser.*, **154**: 1958) dealt with post-graduate training in the public health aspects of nuclear energy; and the other (*Wld. Hlth. Org. techn. Rep. Ser.*, **155**: 1958) examined the introduction of radiation medicine into the undergraduate curriculum. It would be logical to take the basic undergraduate course first.

As we become increasingly aware of the load already placed on the shoulders of the undergraduate student in medicine, nursing and the allied professions, we can find a place for the new knowledge only by some revision of the existing curricula; and in each case we must ask ourselves what will have a real value for the student in later professional life. To take as an example the medical course, on what matters of public concern and anxiety will the family doctor be expected to give advice and establish confidence? It is clear that in the preliminary period of education, some further emphasis must be laid on the concepts of fundamental science. Basic physics at this stage is a rich source for illustration, and, as the Expert Committee pointed out:

"The recent advances in nuclear physics are among the most revolutionary developments in thought that have been experienced by our generation, and a general acquaintance with them must therefore form an essential part of the education of every cultured person."

The Expert Committee prepared a brief schedule of topics recommended to be included in a pre-clinical course in physics. I lay some stress on this because, with minor variations, it would apply to training for other professional workers in the health field, especially when reinforced by practical illustrations suited to the individual professions such as nursing or sanitary engineering. The principal items are:

(i) the nature of radiations, and their relation to the structure of matter;

(ii) physical, chemical and biological effects of radiations, with particular reference to their technical applications in medicine and their injurious effects on somatic and germ cells;

(iii) detection and measurement of radiation; instruments and units;

(iv) natural sources of radiation (internal and external);

(v) artificial sources of radiation—apparatus for the emission of ionising radiation, and radioactive isotopes;

(vi) chemical properties of some elements with important radioactive isotopes, such as germanium, rubidium, strontium, xenon and caesium, whose stable isotopes are unfamiliar in biochemistry;

(vii) the principal applications of radiations in biology and medicine;

(viii) the more important radiation hazards, aspects of protection and prevention.

When training passes into the more strictly professional phase, a progressive deviation in the material of teaching may take place. Some subjects are of such universal importance to an understanding of the problem that they should be dealt with specifically. The

most important of these are genetics, radiation physiology and relevant aspects of biochemistry.

It is not for me to go into the clinical period in the teaching of medicine or nursing, or the corresponding advanced training of the sanitary engineer. Enough to say that both the personal and the genetic risks of radiation must be emphasised, and that a sound practical knowledge of protective measures must be fully understood. The essential point is that the members of the health team will be asked questions by a public brought up on the fodder provided by the daily newspapers, television and cinema. What is more, the student must be taught by illustrations which will enable him to "go one better" than the lay press in meeting public needs and allaying unnecessary fears. If his own teaching is skilfully reinforced by visual aids, experiments and visits to actual installations, he will be well equipped to speak with authority.

We ought not to underestimate the critical force of public opinion, even in an age which takes so many of its emotions in a kind of two-dimensional way. When it comes to a crisis, the ignorant will be found out, and the people will follow the lead of knowledge.

When we come to the post-graduate period, the problem of training is in some respects simpler. Before very long all students who have passed through professional training in the field of health will have acquired some general knowledge of radiobiology. In all branches of public health study they will be equipped to handle the more strictly public health challenge of radiation.

Four Instructional Levels

In post-graduate teaching the Expert Committee recognised four instructional levels. At each level they considered the subjects under five main headings: physical principles; biological principles; applications of radiation, radioisotopes and nuclear energy; hazards; and protection. It would be well worth while for those who are concerned with planning courses to examine these four levels, as they can be applied to staff members of a health department who have difficult responsibilities and different backgrounds of experience.

At the first level there would be an *orientation course*, suitable as a basic course for all types of health personnel who do not necessarily have specific duties to perform in regard to radiation. It would consist of about six to eight lectures, linked as far as possible with other teaching subjects. Emphasis would be laid on the practical aspects of hazards and protection. This course would be designed for industrial and general nurses; hospital administrators; health educators; and sanitarians.

At the second level an *introductory course* would be appropriate as a basic curriculum for industrial medical officers, sanitary engineers and inspectors, and biostatisticians; and as a further course for medical and veterinary officers who were not expected to have any special responsibility in radiation. It might occupy about 60 hours if given during full-time graduate studies, or two weeks if designed as a special concentrated course. In addition to lectures, opportunity would be given for group discussions and practical exercises. Instruments and equipment would be shown, as well as specially designed films.

The *advanced course* would be designed for medical officers with a definite responsibility in radiation and atomic energy activities; industrial and sanitary engineers; physicists in the health and hospital services; and biostatisticians. The duration would be approximately 120 hours if given during regular graduate studies or weeks as a special concentrated class. Both academic and practical work would be included, and arrangements made for field visits.

The *specialist course* is much more high powered and is intended for persons with professional backgrounds in science, engineering or medicine who will have special responsibilities in the radiation field or atomic energy. The Expert Committee did not recommend a specific duration for the course, but it clearly had in mind at least an academic year of full-time graduate studies. A specialist course of this kind would be quite unsuitable for short-term concentration.

Special Needs

Another feature of the specialist course is that it might have to be planned on a more or less individual basis to meet the special needs of restricted groups. Some of these would undoubtedly have to concentrate on experimental work in their own spheres, and both laboratory work and practical training would have to be arranged accordingly.

It should be borne in mind that post-graduate training cannot be considered in isolation from either undergraduate training or the general education of the public. All the members of the staff of a public health department are likely to have to deal with *some* aspect of radiation or nuclear energy. Public health workers will also be responsible in varying degrees for making recommendations to their authorities on the health and safety aspects of ionising radiation and radioactivity.

The health officer will be responsible for assessing the effects of radiation exposure on the general population not directly concerned with handling and using radioactive materials. He may well be the first source of information and advice on the health and safety of workers engaged in work connected with radiation. Secondly, the health officer will be called upon to advise on the safe use of radiation, including x-rays for both diagnosis and treatment, and in its use in industry. The sanitary engineer will also be concerned with levels of radioactivity encountered in the environment. Other members of the public health team will be charged with the duty of public information and, it may be, soothing the apprehensive and over-excited.

For all these purposes, it is essential that the post-graduate training of the public health officer should pro-

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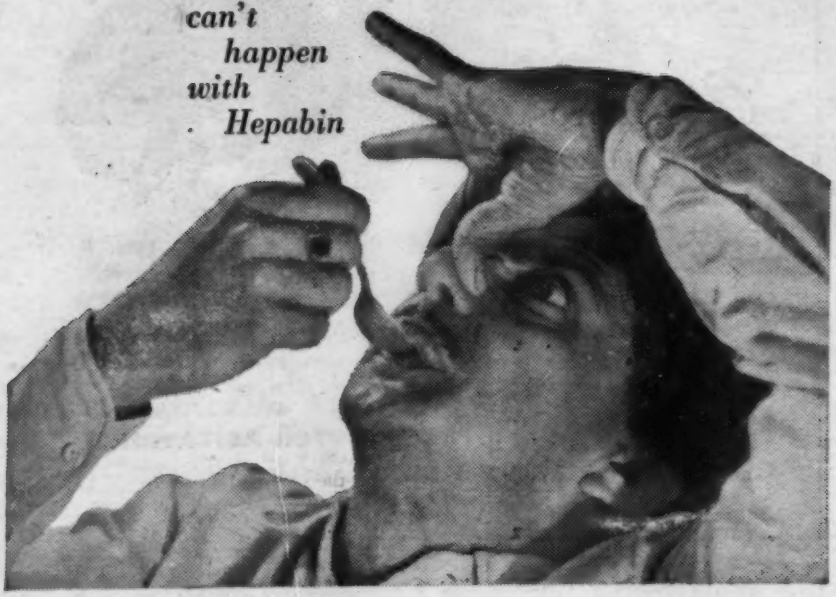

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vide information on which he can teach and act, and deal with emergencies. He must be an authority on all the community implications of atomic energy, and no less on the negative aspects, such as protection of hospital staffs, of industrial workers, and of the community as a whole in the event of local accidents or major catastrophes.

In view of all these considerations, the comprehensive training of the health officer and the potential members of his team becomes of outstanding importance. The principal members of this team are the medical officer of health, the public health nurse, the sanitary engineer and colleagues in environmental sanitation. This does not cover the list of those who require training in radiation health; one immediately thinks of industrial hygiene workers, veterinary public health officers, and laboratory and other technical personnel. There are many other grades of health workers who have important duties in this connection. One should make special reference to those whose function it is to educate public opinion, to provide authentic knowledge and at the same time help to dispel irrational anxieties. This is not merely a technical function, but a problem of supporting moral and spiritual strength.

Conclusion

In this short paper, I have tried simply to do two things, and much of the detail of teaching in radiobiology has had perforce to be omitted. First, I have attempted to give some indication of the constantly enlarging interests of the World Health Organisation in the entire sphere of radiation and the health aspects of atomic energy. I have also tried to make it clear that my subject has been limited to the health aspects of teaching and practice, without attempting to range widely over the general field. In the second place, I have tried to show the essentials of a series of teaching programme recommended by our experts. Their work is very recent and their standing, both in public health and in the medical aspects of radiation and atomic energy, is very high indeed.

We in the World Health Organisation rely a great deal upon the system of bringing together, for a period of perhaps a week or ten days, groups of experts in specific subjects. We make special efforts to get their advice on the subjects of tomorrow rather than the findings of yesterday. I know well that you all realise the great and urgent importance of studies of this kind, and the need for all members of the public health profession to be kept thoroughly informed on pertinent developments in the rapidly moving health sciences. It is only in this way that they can win confidence and prevent complacency on the one hand, and the equally dangerous panic on the other.

The whole situation has been summed up very well by Shakespeare with his usual masterly touch:

If it be now, 'tis not to come; if it be not to come, it will be now . . . . the readiness is all. (Hamlet, V. ii. 232).—*WHO Chronicle*, **14**: 370, October 1960.

NOTES AND NEWS

New WMA Consultant

Dr. Louis H. Bauer, Secretary General of the World Medical Association since 1948, will become Consultant to that organisation on January, 1961. Dr. Bauer has been closely associated with organised medicine since 1929. Dr. Heinz Lord of Barnesville, Ohio, will succeed Dr. Bauer as Secretary General of The World Medical Association.

The XVth General Assembly of The World Medical Association will be held in Rio de Janeiro, Brazil, September 15-20, 1961.

Rehabilitation of Leprosy Sufferers

Early detection and treatment of leprosy may prevent the deformities which brand the leprosy patient for life and make it difficult for him to be accepted in society as a normal member of the community even after he has been cured. This opinion was expressed at the Scientific Meeting on Rehabilitation in Leprosy held in Vellore in the last week of November (21-29 November).

Sponsored by the World Health Organisation, the Leonard Wood Memorial (American Leprosy Foundation) and the International Society for the Rehabilitation of the Disabled, the meeting was attended by top-ranking scientists and plastic and orthopaedic surgeons from a number of countries including India, Japan, Mexico, Philippines, U.K., and U.S.A. The World Health Organisation was represented by Dr. R. H. Bland, Assistant Director, Health Services, Services, South East Asia Regional Office, New Delhi, and Dr. J. Gay Prieto, Chief of Leprosy Section, W.H.O. Headquarters, Geneva. Dr. James A. Doull, Medical Director of the Leonard Wood Memorial, Washington, U.S.A., was elected Chairman.

A report adopted by the meeting said that while an exact estimate of the prevalence of leprosy was impossible at present, 10 million cases in the world was probably a conservative estimate. Of these, fewer than 5 per cent could be accommodated in existing institutions. The vast majority were living in their own homes and probably not more than 20 per cent were receiving treatment of any kind. The second WHO Expert Committee on Leprosy had estimated that 25 per cent of all leprosy patients suffer from some degree of physical disability.

As a means of accelerating progress in rehabilitation the meeting strongly urged that leprosy be studied and treated along with other diseases in centres where a wide range of medical scientists was available. Leprosy research should no longer be carried out only in institutions confined to leprosy and by leprosy specialists who do not have the assistance of basic scientists and experts in other fields. In addition to strengthening leprosy research this would have a great psychological advantage. It was felt that as long as the medical profession continued to treat leprosy separately from all other diseases the public could hardly be expected to believe that it was not "a disease apart".

It was recommended that survey be undertaken not only to find out the extent of the disease but also to

study the manner and time of the onset of deformities and their relationship to the stage of the disease, the treatment given and the occupation of the patient.

The meeting discussed the causes and cures of the various types of deformities—of hands, feet, face and those resulting from bone changes. Facial deformities such as collapse of nose, loss of eyebrows and sagging face were recognised to be of great significance in the rehabilitation of patients. The scientists agreed that these deformities were to a large extent preventible and in advanced cases much could be achieved by corrective surgical treatments.

The meeting stressed the need for large-scale educational and propaganda campaigns to inform the public about the facts of the disease. It was felt that widespread and deep-rooted prejudices with regard to leprosy formed the greatest single barrier to rehabilitation. The public should be educated to appreciate the fact that leprosy is curable and that the deformities which remain after cure do not necessarily mean that the disease is still active.

Rehabilitation agencies in various fields were urged to include leprosy patients in their programmes. It was felt that the experiences of these agencies in combating prejudice concerning physical disability and in mobilising professional and public understanding could be a great asset in developing future leprosy programmes.

Equal stress was laid on the education of the patient himself. He should know what precautions to take and what routines to follow to avoid getting deformities. Rehabilitation should begin when the disease was first diagnosed. Therefore, the doctor or para-medical worker in the field must help all patients to adjust themselves both to the limitations imposed by the disease and to the expectancy of returning to full and normal life. It was felt that no rehabilitation programme could be a substitute for this basic education given by medical advisers. The great majority of patients should complete their rehabilitation with no other outside help and without admission to any institution.

Treatment of Tuberculosis Patients at Home

A year of follow-up studies in a tuberculosis control project in Madras has strengthened the earlier conclusions that "the results of home treatment by drugs approach sufficiently closely to the results of sanatorium treatment to suggest that it is appropriate to treat the majority of patients at home."

The studies—reported in the *Bulletin of the World Health Organisation*, Vol. 23, No. 4-5 (1960)—have been carried out by the Tuberculosis Chemotherapy Centre, Madras, under the joint auspices of the Indian Council of Medical Research, the Madras State Government, the World Health Organisation and the British Medical Research Council.

When the original report was issued, a year ago, it was stated that further information was needed on several points. Chief among them were (1) does the treatment of TB patients at home, instead of a sanatorium, expose their families to any special risk of contracting the disease? And (2) how would the two groups of patients (home and sanatorium) respond to a second year of treatment?

Question (1) is answered by a report on the family contacts of the patients admitted to treatment either at home or in sanatorium.

The number of cases of tuberculosis developing in the families of patients treated at home was found to be no greater than the number in the families of patients in sanatorium.

Furthermore, serial tuberculin tests performed during the year did not yield evidence that more tuberculosis infection occurred in the contacts of home-treated than in the contacts of sanatorium-treated patients.

Of 26 cases of active tuberculosis which developed among contacts 17 were attributed to infection by the index (original) case before the start of treatment. Further, 15 of the 26 cases were under the age of five years: 21 of the cases were under the age of ten.

The report concludes that the major risk to contacts during the year resulted from exposure to the index case before diagnosis. Contacts of home-treated patients were not exposed to any special risk of contracting the disease as compared with the contacts of those treated in sanatorium but it must be emphasised that the patients were given effective treatment.

Question (2)—How do home and sanatorium-treated patients respond to a second year of treatment?—is answered in a further report, which, after recording exhaustive tests, states:

"It is concluded that, at two years, there was very little difference between the patients in whom the disease had reached a stage of quiescence following a year of treatment at home and those who had reached that stage after a year of the same treatment in a sanatorium".

International Congress on Diseases of the Chest

In early September of 1960 some 2000 physicians from all parts of the world and their families gathered in Vienna to attend the 6th International Congress on Diseases of the Chest. The American College of Chest Physicians in Chicago is the initiator of these Chest Congresses, held in some medical centre once in two years.

Professor Karl Pellinger, head of the 2nd Medical Clinic of the University of Vienna, acted as the President of the Congress. The Secretary General was Professor Anton Sattler.

From the very outset, the Organising Committee was in agreement that the scientific programme should deviate from, or at least supplement the routine Congress pattern of endless lectures. Stress was on the coordination of geographically divergent ideas through the free exchange of opinion between specialists from different countries on very specialised problems, including perhaps budding, but not yet print-ready ideas on revolutionary new methods of treatment. With this in mind, the scientific secretary, Dr. P. Mlczoch, organised 42 colloquia on very specialised subjects such as e.g. the therapeutic problems of cor pulmonale, surgery and cardiac arrest, hypothermia, psychosomatic problems

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in asthma bronchiale, clinically useful classification of emphysema, alcohol and tuberculosis, the transplantation of organs, etc. No papers were admitted, only prearranged short discussion remarks, followed by a free discussion from the floor.

The participants from India were numerous and that scientifically very interesting contributions came from attending Indian physicians. Ranging prominently among these were Professor Raman Viswanathan from the Chest Institute of the Delhi University, and Professor Yudhvir Sachdeva from Amritsar. Professor Sen from Calcutta, unfortunately, could not come to Vienna at the time of the Congress, but he arrived there two weeks later and still found good opportunities to converse with Austrian colleagues.

An attractive entertainment programme which was gone through, including a reception by the Lord Mayor of Vienna in the Vienna City Hall, marked the hospitality of modern-day Vienna.

At a meeting of the Executive Council of the American College of Chest Physicians during the Congress it was decided that the next Congress should be held in New Delhi in 1963, with Professor Viswanathan acting as the Secretary General.

Beit Memorial Fellowship

The Beit Memorial Fellowships to promote the advancement by research of medicine and allied sciences in their relation to medicine are available to Indian nationals. The next election of junior fellows for the award of such fellowships will take place in May 1961. Junior Fellowships have successive values of £900, £950 and £1,000 for three years. Candidates must have taken a degree in a faculty of a University approved by the Trustees in Her Majesty' Dominions, Protectorates and Mandated Territories, India, Pakistan and the Republic of Ireland, or a medical diploma registrable in the United Kingdom. Elections to Junior fellowships are rarely made above the age of thirty-five years. Applications from candidates must be received not later than the 1st February, 1961. Candidates must submit evidence that they can be given accommodation in the Departments where they propose to work, which must be either in Great Britain or Ireland. Forms of applications and other detailed information may be obtained from the Secretary to the Government of India, Ministry of Health, New Delhi.

Organism and Human Cells in Space

Human cells sent into orbit around the earth only recently in an American satellite are reported to have survived a radiation bombardment caused by a massive gas explosion on the sun, by scientists at San Antonio, Texas, U.S.A. The cells were in the capsule of the Discovery XV satellite which travelled a million miles in space between November 12 and 14.

The capsule was recovered in mid-aid over Hawaii. A similar recovery of the capsule of Discoverer XVIII which also contained human cells and algae was subsequently made.

A nuclear-physicist at the School of Aviation Medicine at San Antonio reported that the capsule of Discoverer XVIII had been subjected to unexpectedly heavy radiation from a solar flare caused by an explosion on the sun of near record size.

The New York Times has reported that living organisms rocketed into space through the radiation belt surrounding the earth had been recovered in a state of apparent death and yet were found capable of revival in a recent test.

The organisms—Neurospora, a form of bread mould—were carried into space on September 19 in a rocket fired from the Navy range at Point Arguello, California, and spent 26 minutes inside the inner Van Allen radiation belt.

Public Health Nutrition Seminar

The seminar of Public Health Nutrition held in Calcutta in November was opened with a discussion on the Role of Human Volunteer Experiment in Solving Nutritional Problems by Dr. John Pemberton of WHO at the All India Institute of Hygiene and Public Health.

Dr. R. N. Chaudhuri, Director, School of Tropical Medicine, Calcutta, discussed the problems and various aspects of malnutrition amongst infants and children. From his own experience, he came to the conclusion that these were mostly due to protein as well as calorie malnutrition. The most vulnerable group was children of the age group 6 months to 3 years.

Shri S. Mukherjee, Anthropologist, All India Institute of Hygiene and Public Health, stressed the influence of various factors, viz., religious and social and taboos on the food habits of the people which in turn is mainly responsible for the malnutrition amongst the various sections of the people and a thorough study of these food habits and their origin was considered very important for improvement of nutritional status.

Dr. N. Bagchi, Associate Professor of Biochemistry and Nutrition, All India Institute of Hygiene and Public Health, described a vitally important 'human experiment' that was being carried out at the Institute. In this country the economic condition of the people and other factors did not permit adequate and liberal consumption of milk by the children and as a result gross malnutrition was prevalent amongst the children of low income group. A practical approach to solve this problem would be to introduce protein-rich food which was easily available within the reach of the people, and one such item of food is Bengal gram. He disclosed that the children of the age group of 1-5 years were fed palatable food prepared from Bengal gram and compared with similar food made out of skim-milk powder. A small group of children was also kept as control. After a study of 36 months, it was observed that there was no significant difference in the improvement of the nutritional status of the children in the two experimental groups.

The afternoon session took up the subjects of Epidemiology of Mental Diseases and Psychotherapy in Children. Dr. Pemberton gave an elaborate lecture of the position of mental health in Great Britain. He dealt with problem families in U.K. and tried to bring out

the epidemiological factors leading to such conditions. According to him, 5000 committed suicide in U.K. annually and of them over 3,000 were male. He stressed on the point that the epidemiology of mental illness was rather uncertain.

Dr. S. Banerjee, Professor of Psychiatry, R. G. Kar Medical College, Calcutta, in presenting the condition of mental health in India pointed out that mental health had been neglected in this country although health and mind were inter-related. He stressed on the need of a survey to be conducted by All India Institute of Mental Health to assess the magnitude of the problem in India.

Dr. N. C. Mukherjee of the All India Institute of Hygiene and Public Health dealt with the mental tests and psychotherapy in children.

Earlier Shri S. Ghosh, Inspector of Smoke Nuisance, Government of West Bengal, spoke on Smoke Nuisance after the subject was initiated by Dr. John Pemberton. Shri Ghosh stated that combustion of fuel was linked up with the process of normal urban life and therefore some nuisance was a problem in Calcutta. Factors like incomplete combustion of fuel in the furnace and inefficient utilisation of the energy liberated were responsible for this nuisance. The Smoke Nuisance Act of West Bengal tried to control it through control measures like erection of chimney in accordance with approved plans and fitting of mechanical stokers for large furnace.

Andhra Rural University

The Andhra Rural Education Board, Visakhapatnam, invites suggestions from members of the medical profession for the proposed courses for the Andhra Rural University. The courses are to meet the needs of the rural people. The post-graduate courses for medical personnel comprise training in other systems; practical training; short-term courses in ophthalmology, obstetrics and gynaecology. Those for health workers include courses for health visitors, midwives and nurses. Comments are to be sent to the Secretary at Chodavaram, Visakha District, Andhra Pradesh.

Conference of Pathologists

Presiding over the 11th annual conference of the Indian Association of Pathologists on 1-12-60 in Hyderabad, Prof. Edward Gault, of the Christian Medical College, Vellore, stressed the need for all-India unification of standards for post-graduate training. There were indications that it would be achieved in the near future, he said. There was a great need for observations, recording and deduction in the study of disease in India today. In this age, when there was too great a tendency to think in terms of examinations only, they should cultivate that association of experience with youth and enthusiasm.

Inaugurating the conference, Srimati Masooma Begum, Minister for Social Welfare, hoped that the State would be in the forefront of medical education, medical care and public health services.

Dr. K. N. Rao, Chairman of the Reception Committee, in the course of his welcome address, said the time had come for a national council of medical scientists to discuss, evolve, plan and to execute the scientific progress in medicine and also to raise the standards of medical care in the country.

Conference of Physiologists and Pharmacologists

Inaugurating the sixth annual conference of the Association of Physiologists and Pharmacologists in Hyderabad on December 1, 1960, Prof. M. S. Thacker, Director-General of the Council of Scientific and Industrial Research, said that the official "procedures and drills" which have been prescribed, were retarding instead of helping the cause of science.

Prof. Thacker said that his survey of research development in this country and the progress made in the fields of scientific and technological education, revealed a number of lacunae.

Speaking of the importance of biological sciences, he said that only during the last three or four years was a real impetus given to them. On the recommendation of a committee, appointed for the purpose, they had taken a decision to open in the next five years a Central Institute of Biological Sciences.

In conclusion, Prof. Thacker assured the medical scientists that in their endeavours they would get the fullest support of the Ministry of Scientific Research.

Mr. D. S. Reddi Vice-Chancellor, Osmania University, who presided, said that the two functions of the University were the dissemination of knowledge and advancing the frontiers of knowledge by research. Both these were inter-related. A good teacher could not be a good teacher unless he was also interested in research.

Earlier Dr. K. N. Rao, Director of Medical Services, welcomed the decision of the conference of Vice-Chancellors of South Indian Universities that English should continue to be the medium of instruction for professional and technological studies.

Prof. R. B. Arora, Honorary General Secretary, Association of Physiologists and Pharmacologists, read a report. Dr. M. Y. Ansari, Principal, Gandhi Medical College, proposed a vote of thanks.

J. C. Bose Memorial Lecture on Cancer

A survey of the geographical pathology of cancer in India has yielded valuable information regarding the probable aetiology of intraoral, breast and certain epithelial cancers, peculiar to the people of different States or communities in the country, said Dr. V. R. Khanolkar, Director, Indian Cancer Research Centre, Bombay, delivering the 22nd Acharya Jagadis Chandra Bose memorial lecture at Bose Institute, Calcutta, on Nov. 30, 1960.

Dr. Khanolkar dealt with some aspects of the cancer problem, which had a bearing on the work being done at the Centre for the last several years. Cancer was a world problem, and a study of naturally occurring human cancers in different people had proved a fruitful field of research by providing information not obtainable from animal experiments.

The speaker said that a vital aspect of the cancer problem was the treatment of the disease. Surgery and atomic radiations were effectively employed in treating certain types of cancer. They were of limited value due to various causes, such as wide-spread dissemination of the primary growth or radio resistance of some tumours.

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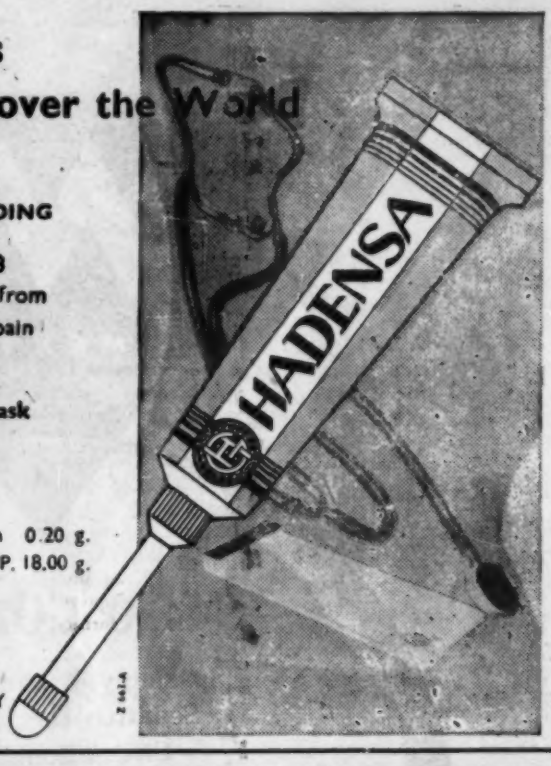

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The latest approach to the treatment of cancer is chemotherapy. The efficacy of alkylating agents and antimetabolites used in anti-cancer drugs was evaluated. A new approach to the treatment of cancer, which was being followed at the Indian Cancer Research Centre, was described by Dr. Khanolkar.

Medical Education Association Formed

Inaugurating the Indian Association for Advancement of Medical Education, in Hyderabad on December 4, 1960 the Union Minister for Health, Dr. D. P. Karmarkar, made an emphatic plea for multiplying medical and health personnel at the lower level and said unless this was done "we will not be able to serve our country".

Welcoming the formation of the Association, Mr. Karmarkar opined that all did not seem to be well either with the curriculum or methods of teaching or examination in medical education. He would like this matter to be inquired into.

Dr. A. Lakshmanaswamy Mudaliar, Vice-Chancellor of Madras University, who has been chosen the first President of the newly-formed Association, in his presidential address said he was glad the Association had been sponsored by the Indian Council of Medical Research as he was of the firm opinion that research and medical education should go together. He regretted that there had not been the close liaison that should exist between the medical institutions of the country and the research projects that might be undertaken. Medical education and research had not always gone side by side in close harmony and to the mutual advantage of both. Speaking of the role the Association could play, he said there might be some misgivings in some quarters that the Association was superfluous as there were other statutory bodies recognised for the maintenance of standards in medical education. There were, he stressed, a variety of topics of great importance and interest which at prestnt could not be tackled by any other means.

Dr. Mudaliar added : It should be realised that unless there was an organisation which would, in a sympathetic and understanding manner, go into the many problems of medical education—under-graduate, post-graduate and research—and give its frank opinion in assessing the merits of various medical institutions, the mere fact that certain statutory bodies recognised these institutions for purposes of enrolment of the alumni into the medical register would not lead to that level of improvement which would entitle India to be recognised as one of the great centres of medical education.

CORRESPONDENCE

National Formulary of India Corrigenda

SIR,—The National Formulary of India was published in May, 1960. Our attention has been drawn to certain mistakes in it which have inadvertently crept in. A list indicating the corrections is prepared and is now under print. Since the printing may take some time it is felt that it would be desirable to give immediate publicity to the corrigenda list. I am enclosing herewith a copy of the corrigenda list and it will be greatly appreciated if the list is published in your Journal. I am etc.

New Delhi — S. K. BORKAR, Drugs Controller (India)

CORRIGENDA

Page	Line	For	Read
85	4	0·75	7·5
92	6	0·16	16 mg.
93	1 from bottom	12 ml.	0·12 ml.
95	Delete last two lines		
96	After line 5 add a line Syrup.... 10 ml.		
97	4 from bottom	130	180
107	2 from bottom	on	or
116	9	0·15	0·25
119	1	5	6
122	23	60	100
122	11 from bottom	Delete per ml.	
126	7	Delete chloride	
127	14	niacin	niacinamide
127	14	1000	100
133	11	50	25
136	4	0·3	0·03
137	7 from bottom	0·6 mg.	0·6 g.
142	4 from bottom	6·9	0·69
143	29 to be followed by : It should be freshly prepared		
153	6 from bottom	2·5	0·5
155	1	Delete the word 'compound'	
155	2	5	1
162	9 from bottom	1·24	1·25
162	5	1·3	1·5
163	14	After Ammonia add I.P.	
163	18	0·5	0·3
169	9	150	160
174	9	1	0·5
175	4	40	60
178	1	After Cyclizine add Hydrochloride	
178	4	After Cyclizine add Hydrochloride	
182	14	6	21
197	14 from bottom	$\bar{B}$,	B_1
197	1 from bottom	Delete Hydrochloride	
198	1	Delete Hydrochloride	
204	9 from bottom	7·7	7·6
205	6 to be followed by : Dose 2 to 4 ml.		
206	11 from bottom	0·06	0·03
207	6 from bottom	0·06	0·6
208	5	120	150
208	9	0·076	0·08
209	11	0·2	0·02
211	13	106	166
242	9	68·5 mg.	68·5 g.

The National Formulary of India compiled by the National Formulary Committee, 1960, Government of India, Ministry of Health, New Delhi, was reviewed in this Journal (**35** : 478, 1960).—ED., *J. Indian M. A.*

REVIEWS

Lymphocytes and Mast Cells—By M. A. Kelsall and E. D. Crubb; 1959, Ballière, Tindall & Cox Ltd., London, 6"×9", pp. xvi+399; figs. 31; 64s.

This monograph is the outcome of extensive review of the various ideas and experimental results prevailing in connection with some of the important connective tissue cells such as the lymphocytes, plasmacytes and mast cells. Materials have been collected, to form a working hypothesis for these from various fields of biological sciences including anatomy, embryology, biochemistry, cytology, enzymic chemistry, pharmacology, medicine, etc. These cells have been collectively termed trephocytes because of their primary importance in intermediary metabolism related to growth, normal and abnormal. Their general and primary function is directly related to the metabolism of other fixed cells, by synthesis, storage and transport of nucleoprotein besides specific functions as in the case of mast cells which control the passage of nutritive material from capillaries to the individual cells by synthesis and release of histamine and heparin affecting capillary permeability and reducing the viscocity of the collagenous ground substances of connective tissues. The book is divided into twelve chapters, the first chapter being devoted to general principles and actions of trophocytes and the last to inflammation, wound healing and also the effect of starvation, cortisone, etc., on these cells and their activites. The book is packed with valuable information supported by excellent references to literature and will be very useful to teachers and research workers irrespective of their primary fields of interest.

P. Bose

Leukaemia : Research and Clinical Practice—By F. G. J. Hayhoe, M.A., M.D., (Cantab.), M.R.C.P. (Lond.); 1960, J. & A. Churchill, Ltd., 104 Gloucester Place, London, W. 1; 9¼"×7¼", pp. vii+335; 12 colour plates, 196 monochrome illustrations; 80s. net.

In the preface the author very aptly states "the rising incidence in leukaemia and its possible relationship to background or occupational exposure to ionizing radiations in this atomic age has brought the disease increasingly before the public eye." Leukaemia, as one of the most important problem-diseases has still been baffling the skill of the pathologist, the haematologist and the clinician in general. Dr. Hayhoe has done a remarkable service in presenting the current concepts of the disease in this book, so conveniently divided into four chief sections, satisfying the needs of different workers in the field. The chapters on clinical aspects and general principles of therapy have been very lucidly dealt with. The role of plant derivatives (vinca-leucoblastin) derived from *Vinca rosea* Linn. has not been mentioned in this book perhaps due to the fact that the material has not yet established its value as an aid to therapy.

The reviewer fully agrees with the statement of the author : "the fact that the present methods of treatment are purely palliative and do not prolong the life-span lends a sharp spur to research efforts in this direction". This book has the stamp of an admirable production and as such is certain to find a place in the book-shelf of clinicians, research workers and studetnts.

G. B. Sinha

Modern Concepts of Thyroid Physiology—Annals of the New York Academy of Sciences, Volume 86, Art. 2, April 23, 1960; Editor, Otto v. St. Whitelock; Published by the Academy, 2 East 63rd Street, New York 21, U.S.A.; 9"×6", pp. 311.

It has been a pleasure to see all possible recent work in the line of thyroid physiology assembled in this volume.

All the papers are no doubt valuable, but the reviewer cannot do without saying a few words about some which have specially attracted his interest. In Thyroxin and Swelling and Contraction in Mitochondria by Lehninger, the author has gathered evidence regarding the role of thyroxin in connection with electron transfer system. Mention should also be made of Effect of Thyroid Hormones on Adrenal Steroid Metabolism by Tomkins *et al.* The authors have helped us through their recent work to go ahead in this line of research. The thoughtful discussion with experimental evidence in the background of Rose *et al* in their paper of Regulation of T. S. H. Release has added strong support to the suggestive theory of the role of hypothalamus on the regulation of T. S. H. release from the anterior pituitary. Their experimental evidence supporting the theory of transport of some neurohormone through the hypophysial portal system has supplied an extra inspiration to the investigators in this field.

S. R. Mukherjee

Chikitsa Jagat (A medical monthly in Bengali)—Edited by Dr. A. D. Mukharji, 32nd Anniversary Number on Advances in Medicine, October 1960; P 79, New C. I. T. Road, Calcutta 14; 9½"×7¼", pp. 88; Rs. 2·00.

The *Chikitsa Jagat* has a record of service to the medical profession—it has through the last 31 years of its existence created a tradition of consistently catering medical literature and disseminating uptodate knowledge of scientific medicine particularly amongst doctors practising in remote villages.

This the 32nd anniversary number epitomises in the main the progress in different branches of medicine besides dealing with various aspects of diseases and treatment maintaining at the same time some of the regular features of the publication. The contributions are from distinguished practitioners and academicians and the articles, rich in materials, cover a wide range. The collection in this issue, numbering about 20 items, is an eloquent testimony to the enterprise and resourcefulness of the journal. There is a stamp of excellence both in the presentation and production. The Editor and the publisher deserve congratulations for bringing out this anniversary number which is certainly worth more than the price at which it is offered.

S. K. S.

SUPPLEMENT

Journal of the Indian Medical Association

Vol. 36 No. 1 CALCUTTA January 1, 1961

BRANCH NOTES

ARARIA BRANCH—The annual general meeting of the branch was held on 17-11-60. Dr. L. M. Kundu presided. Twelve members were present. Dr. J. N. Srivastava gave a talk on Malaria Surveillance.

Office-bearers for 1960-61 were elected with Dr. L. M. Kundu as president; Dr. (Mrs.) L. P. Mukherjee as vice-president; Dr. T. N. Saha as hony. secretary; Dr. Md. Siddique as asst. secretary; Dr. B. B. Dasgupta as auditor; Dr. N. Nandi as representative to the Central Council with Dr. S. Rehman as alternate; Dr. N. G. Bhowmick as representative to the State Council.

BAREILLY BRANCH—The following are the office-bearers of the branch for 1960-61 : President—Dr. C. M. Aga; vice-presidents—Dr. A. D. Bhandari and Dr. P. C. Bhalla; Secretary—Dr. M. K. Goel; Treasurer—Dr. J. S. Garg.

BERHAMPORE (GANJAM) BRANCH—A meeting of the branch was held on 16-11-60. Twenty-six members were present. The meeting by a resolution invited the annual conference of the Orissa State branch at Berhampore in the 1st week of May, 1961. Dr. K. Satyanarayana Rao spoke on Tetanus and Trends in its Treatment.

COIMBATORE BRANCH—A monthly meeting of the branch was held on 22-11-60. Sixty members were present. Dr. S. Sundareswaran presided. Two new members—Dr. C. R. Subramanian and Dr. T. K. Rithuparnan—were introduced to the meeting, Dr. V. Govindan Nair delivered a lecture on Modern Conception of Syphilis.

DANTAN BRANCH—A general meeting was held on 1-10-60. Dr. R. N. Chakraborty presided. Ten members were present. Office-bearers for 1960-61 were elected with Dr. K. Dutta as president; Dr. R. Mahapatra as vice-president; Dr. J. Bhowmick as hony. secretary; Dr. S. L. Saha as treasurer; Dr. D. Mukherjee and Dr. R. N. Chakraborty as members of the Provincial Council. It was decided to hold meetings more frequently and to arrange a rural medical conference. Cholaemia was discussed at the meeting.

DELHI STATE BRANCH—The Delhi Medical Association views with alarm and dismay the recent practice of the Central and some of the State Health Authorities of declaring obvious epidemics of cholera as outbreaks of gastro-enteritis. Scientifically this practice is utterly wrong, deceives the public and prevents it from taking timely precautions.

Gastro-enteritis is merely a set of symptoms which may be due to multiplicity of causes. However, when cases with these symptoms occur in an epidemic form the cause must be specified to enable health authorities to take effective measures to control it. No epidemics of this type have been known to occur anywhere in the world, including our country, due to any infection other than that of *Cholera vibrio*.

The Union Health Minister addressing the Rajya Sabha on August 30, 1960 stated that in U.P. during the week ended August 13, 1960, there were 2,933 attacks with 1,033 deaths. He asserted that all these were not necessarily cholera cases because *Cholera vibrio* was not found in all the stools. This reasoning is utterly fallacious in the face of the fact that a number of cases occurred in the same house and some were declared as cholera and others as acute gastro-enteritis on the basis of positive or negative isolation of *Cholera vibrio*.

It is well known that *Cholera vibrio* is a very unstable organism and its successful isolation depends on the competence with which samples of stools and other dejecta are collected and examined. Careful investigations have shown that though *Cholera vibrio* can be isolated with good laboratory facilities by competent workers in as many as 80 per cent cases, yet ordinarily *Cholera vibrio* is isolated in only a small percentage of cases when the examination is conducted in a haphazard manner by inexperienced workers.

It must also be remembered that laboratory findings are an aid to diagnosis, but they do not replace the clinical findings. After all, *Cholera vibrio* was discovered only 76 years ago, but cholera as a clinical entity has been recognised since 7th century B.C. as described in Sushruta, and epidemics of cholera have been recorded in India since that time. Anyhow, from 1877 to 1941, even though the system of reporting was none too good, there was not a single quinquennial period which was free from cholera epidemics which claimed on an average as many as 150,000 to 450,000 deaths a year, and no cases of this new so-called gastro-enteritis were found. And though nothing very effective has been done to eradicate cholera yet the precipitate fall in the number of cholera attacks and deaths has been reported in recent years due to this unjustifiable practice of labelling it as gastro-enteritis.

If it is a fact that a new infection has invaded India since we gained our liberation which is claiming such high mortality as 1033 deaths out of 2933 attacks in U.P. in the week ended August 13, 1960, the responsibility rests on the health authorities to investigate and specify the nature of the causative agent, devise adequate preventive measures and prepare an effective vaccine against this new infection. Vaccines are very highly specific; to go on giving people cholera vaccine when the infection is not cholera is a medical malpractice, an insult to people's intelligence, and deceiving the innocent public by submitting it to unnecessary injury.

The Delhi Medical Association stresses and intelligence demands that in early cases, even if the cases are not typical and the vibrio is not isolated, they should still be notified as cholera to alert the health authorities and the public so that control measures can be instituted and the epidemic prevented from spreading. This is a time honoured practice of great value and should not be departed from.

It is now well established that cholera can be eradicated and has been successfully eradicated in a number of countries during the last many decades. Our own Government also must stir themselves to carry out effective control measures without further delay to eradicate cholera, and not indulge in this undesirable game of abolishing cholera by giving it a new name.

The Delhi Medical Association demands that this practice of misnaming epidemics of cholera should be prohibited by definite administrative orders until the existence of some alternative infection is established.

The Association expects this administrative action to be taken soon.

FORBESGANJ BRANCH—The annual general meeting was held on 6-11-60 with Dr. A. Niranjan in the chair. Sixty-one members were present. The annual report for the last year was adopted. Office-bearers for 1960-61 were elected with Dr. Niranjan as president; Dr. B. K. Sarkar as vice-president; Dr. M. N. Jha as hony. secretary and treasurer; Dr. R. M. Chaudhury, Dr. D. C. Rakhit as joint asst. secretaries; Dr. P. N. Roy (alternate—Dr. M. N. Jha); Dr. Golab Jha, Dr. N. Mazumdar (Alternates—Dr. K. B. Sahin and Dr. S. Hugal) as central council members. Dr. M. N. Jha spoke on Artificial Pneumothorax and Pneumoperitoneum.

HYDERABAD BRANCH—The annual general meeting of the branch was held on 25-9-60. The following office-bearers were elected for 1960-61 :

President—Dr. V. Gopal Rao; vice-president—Dr. P. Manikya Razu; hony. secretary—Dr. R. B. Davare; hony. treasurer cum jt. secretary—Dr. M. Y. Mirza; hony. jt. secretary—Dr. D. V. Bopardikar; Central Council members—Dr. P. L. Narayan Rao, Dr. Digamber Singh, Dr. A. V. Ramamurthy; Provincial Council members—Dr. B. K. Naik, Dr. C. Sitaramayya, Dr. H. Syed Ali, Dr. M. Vishwanatha Rao and Dr. Digamber Singh.

Prof. O. L. Wade, Prof. Greenfield and Prof. Pemberton of Belfast University were given a reception on 14-10-60, under the presidentship of Dr. V. Gopal Rao. The reception was followed by a talk on Cardiac Output by Prof. O. W. Wade.

KARNUL BRANCH—At the annual general meeting of the branch the following were elected as office-bearers for 1960-61 : Dr. Mrs. Sher Singh as president; Dr. K. N. Dutt as vice-president; Dr. M. Singh Lamba as hony. secretary; Dr. Jaswant Rai as joint secretary; Dr. Sukhdev Singh as provincial representative.

In a meeting held on 6-11-60 with Dr. Major Shadi Ram in the chair, the following cases were shown by the secretary and discussed :

(1) Hyperthyroidism, (2) Myxoedema, (3) Lung Abscess.

KOTHAGUDIUM BRANCH—The annual general body meeting of the branch was held on 28-10-60. Dr. T. C. Ramchandani presided. Eleven members were present. The annual report for 1959-60 was adopted. One president and 3 vice-presidents of the Andhra Pradesh State Branch were elected. Dr. T. C. Ramchandani was re-elected as president and Dr. B. Das re-elected as hony. secretary for 1960-61.

The annual report of the branch for 1959-60 shows that there was no appreciable change in membership during the year. Altogether 5 meetings were held, 3 clinical and 2 general body meetings. The clinical meetings were well attended.

LUCKNOW BRANCH—The annual report of the branch for the year 1959-60 shows a slight decline in membership. Five meetings of the Executive Committee, 2 annual general meetings (one adjourned), one special meeting, 11 scientific and business meetings and 1 emergent general meeting were held during the year. Two medical journals, *B.M.J.* and *The Practitioner* continued to be circulated to the members through a part-time peon. Two members were selected for internship and proceeded abroad. A symposium on Family Planning was organised and an exhibition was held during the Family Planning Week. Other activities included free inoculation against cholera and smallpox, an At Home in honour of Dr. C. O. Karunakaran, the President of the I.M.A.

MADURA BRANCH—A monthly meeting of the branch was held on 22-11-60. Dr. K. V. Mathai of Christian Medical College Hospital gave a lecture on Early Recognition of Tumours of the Central Nervous System. Dr. K. Ramchandran presided.

The members of the branch gave a reception to the new medical graduates of Madurai Medical College on 4-9-60. Dr. B. S. Chellappa presided. Dr. T. V. Sivanandam, president, Madras State Branch addressed the new graduates on IMA's Activities and Achievements. Dr. A. Srinivasan of the Government Hospital, Madras gave a talk on Diagnostic Problems in Mitral Stenosis.

NABADWIP BRANCH—The following office-bearers have been elected for 1960-61 : President—Dr. M. L. Kundu; vice-president—Dr. M. Chakrabarty; secretary—Dr. N. N. Saha; jt. secretary—Capt. A. K. Nath; representative to the central council—Dr. M. L. Ghosh; representatives to the provincial council—Dr. N. N. Saha, Dr. S. N. Mukherjee, Dr. S. Banerjee.

NALGONDA BRANCH—The general body meeting of the branch was held on 6-11-60. Dr. D. Tulsi Das presided. The secretary read the annual report. Office-bearers for 1960-61 were elected with Dr. D. Tulsi Das as president; Dr. J. M. Khan as vice-president; Dr. D. A. Mohan Rao as hony. secretary; Dr. M. C. Narsimhulu as joint secretary and Dr. V. V. Gopal Rao as treasurer. Dr. D. A. Mohan Rao spoke on Colles' Fracture; Dr. P. Kodanda Ramaiah demonstrated a case of Vesical Calculus.

SHAHABAD BRANCH—The annual general meeting was held at Arrah on 4-11-60 with Dr. V. Shanker in the chair. Office-bearers for 1960-61 were elected with Dr. V. Shankar as president, Dr. B. P. Lal as secretary, Dr. S. P. Mukherjee and Dr. (Capt.) Y. N. Singh as joint secretaries, Dr. S. M. Shamin Ahmad as treasurer, Dr. K. K. Saran as auditor, representative for the central council—Dr. Agarwala, Dr. B. M. Sinha, press secretary —Dr. S. P. Mukherjee.

SHILLONG BRANCH—At the clinical meeting held on the 27th October, 1960 at the Khasi Hills Welsh Mission Hospital, the following cases were presented by Dr. P. A. Shave.

(1) A case of Ectopia Vesicae in an adult male patient, aged 30, treated by bilateral ureterocolic anastomosis The complications were discussed, including Hypokalaemia and Hyperchloraemia, and the rare complication of Epididymo-orchitis, which is not described in the literature; (2) A case of local gigantism of one leg in a boy aged 12 years. The diagnosis was discussed. The raised skin temperature of the leg, with lengthening of the limb below the knee, and an arteriogram suggesting an arterial abnormality supported the diagnosis of a congenital arteriovenous fistula. (3) A case of osteogenic sarcoma from the iliac bone in a Nepali boy, aged 15. An unusual feature of the case was the evidence of lymphatic spread to para-aortic and femoral glands, while the lung-fields remained clear. It is also uncommon for a sarcoma to arise from the ilium. Dr. R. A. Hughes then presented the following cases :

A case of Mycetoma spreading *via* the lymphatics from the foot to the groin, causing gross tissue destruction and multiple sinus formation. The condition was not arrested in any way by authorities or fungicidal drugs, but finally responded to treatment with iodide by mouth and a case of Carcinoma of the Oesophagus (jct. middle and lower thirds) treated palliatively by the insertion of a wide-bore polythene tube, but combined oesophagoscopy and gastrotomy. (X-ray shown only). This had been most successful as a palliative measure, giving several months' relief to the patient.

SILIGURI BRANCH—The annual general meeting of the branch was held on 6-11-60 with Dr. T. P. Roy in the chair. Office-bearers for 1960-61 were elected with Dr. G. C. Ghose as president; Dr. K. N. Chatterjee and Dr. A. K. Ghose as vice-presidents; Dr. S. K. Ghose and Dr. K. Dey as hony. joint secretaries; Dr. K. Dey as hony. treasurer.

Nine clinical meetings were held in this year, in which the following 5 papers were read : (1) Toxaemia of Pregnancy by Dr. (Miss) M. Chowdhury, (2) Septic Abortion by Dr. B. K. Roy, (3) Vital Statistics by Dr. P. C. Chowdhury, (4) Nutrition by Dr. R. Bhattacharjee and several case notes by Drs. A. K. Nandy, N. M. Lahiri, S. K. Ghose.

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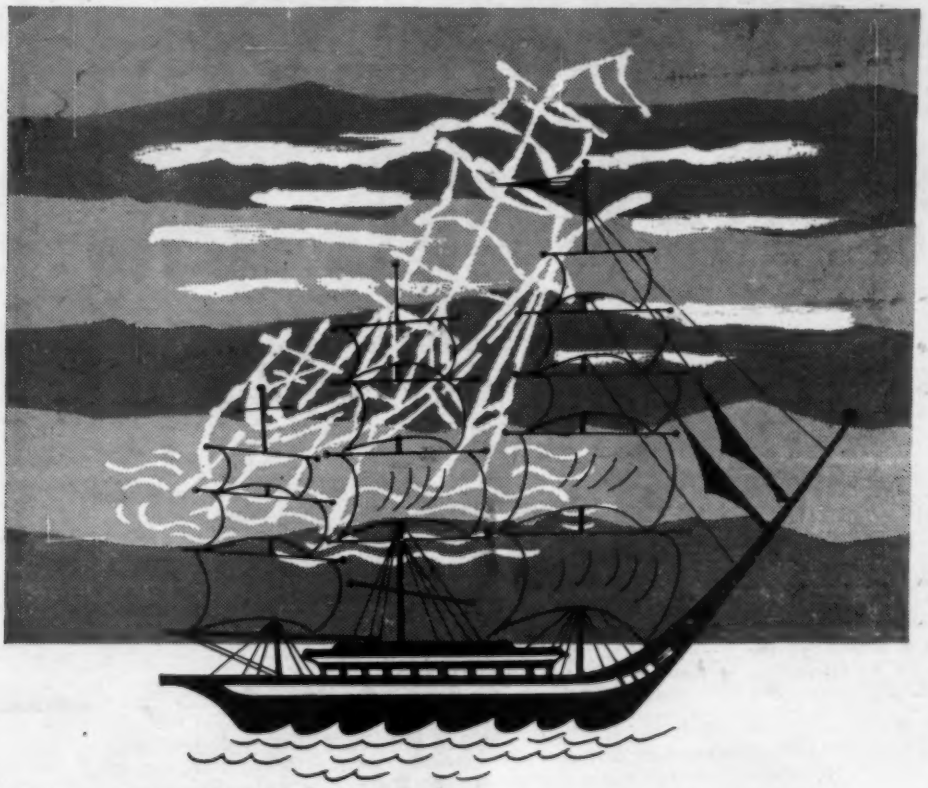

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